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OF

American Physicians and Surgeons

FOURTEENTH TRIENNIAL SESSION

HELD AT

WASHINGTON, D. C.

May 1st and 2nd, 1928

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TABLE OF CONTENTS

	Page
Table of Contents	i
Titles of Papers to be read at Congress	i
Officers of the Congress	iii
Executive Committee	iv
Committee of Arrangements	v
Past Officers of the Congress	vii
By-Laws of the Congress	viii
Minutes of the Fourteenth Session of the Congress	ix
Guests of the Congress	x
Visitors	lxxiv
Recapitulation	lxxvii

THE PRESIDENT'S ADDRESS:—THE DECLINE OF INFECTIOUS DISEASES IN ITS RELATION TO MODERN MEDICINE; BY DR. THEOBALD SMITH 1–18

THE HEART 19–90

Economic Aspects of Heart Disease; by Dr. Haven Emerson 19–38

The Heart in Infection; by Dr. Homer F. Swift 39–61

The Effect of Strain on the Heart; by Dr. Paul D. White 62–75

Comments on the Treatment of Heart Disease; by Dr. James B. Herrick 76–81

Discussion:

Dr. E. P. Carter 82–84

Dr. A. E. Cohn 84, 85

Dr. George Canby Robinson 85, 86

Dr. Henry Sewall 86, 87

Dr. Louis Faugeres Bishop 87, 88

Dr. S. A. Levine 88, 89

Dr. Homer F. Swift 89, 90

Dr. Paul D. White 90

BUSINESS SESSION OF THE CONGRESS 91, 92

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1928

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OF

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1.

This organization shall be known as the

CONGRESS OF AMERICAN PHYSICIANS AND SURGEONS.

2.

It shall be composed of national associations for the promotion of medical and allied sciences.

3.

It shall hold its sessions triennially in the city of Washington, D. C.

4.

The officers of the Congress shall be a PRESIDENT, VICE-PRESIDENTS, A SECRETARY, A TREASURER, AND AN EXECUTIVE COMMITTEE.

5.

The PRESIDENT shall be elected by the Executive Committee, of which he shall be *ex-officio* a member. He shall preside at the sessions of the Congress. He shall deliver an address.

6.

The PRESIDENTS of the participating societies shall be *ex-officio* the VICE-PRESIDENTS of the Congress.

7.

The SECRETARY and the TREASURER shall be elected by the Executive Committee. They shall be *ex-officio* members of the Executive Committee.

8.

The EXECUTIVE COMMITTEE shall be composed of one (1) member from each participating Society and said members shall be elected by the various Societies at the next annual meeting subsequent to the Congress.

It shall be charged with all duties pertaining to the organization of and preparation for the ensuing Congress, including the election of all officers and of a Committee of Arrangements.

9.

The expenses of the Congress shall be divided between the participating Societies in proportion to their membership.

10.

The admission of new associations to participate in the Congress shall be by unanimous vote of the EXECUTIVE COMMITTEE.

MINUTES

The fourteenth triennial session of the Congress of American Physicians and Surgeons was held in the Convention Hall of the Mayflower Hotel, in Washington, D. C., on May 1st and 2d, 1928, according to the programme as ordered by the Executive Committee.

The first session was called to order at 8 P. M., Tuesday, May 1st, by the First Vice-President, Dr. A. McKenzie Forbes of Montreal. Dr. Theobald Smith then delivered his Presidential address on "The Decline of Infectious Diseases and its Relation to Modern Medicine." This was followed by an address in honor of the Semi-Centennial of the American Laryngological Association by the President of that Association, Dr. D. Bryson Delavan of New York City, on "The Founders of Modern Laryngology and Rhinology."

On the following day the meeting was called to order at 2:30 P. M. by the President, Dr. Theobald Smith. The subject to be considered was "The Heart." Papers were read by Dr. Haven Emerson of Columbia University, of New York City, on "The Economic Aspects of Heart Disease"; Dr. Homer F. Swift of the Rockefeller Institute for Medical Research of New York City, on "The Heart in Infection"; Dr. Paul D. White of the Massachusetts General Hospital of Boston, on "The Effects of Strain on the Heart"; and Dr. James B. Herrick of the Rush Medical School, Chicago, on "Comments on the Treatment of Heart Disease." These papers were discussed by Dr. Edward P. Carter of Baltimore, Dr. Alfred E. Cohn of New York City, Dr. G. Canby Robinson of Nashville, Dr. Henry Sewall of Denver, Dr. Louis F. Bishop of New York City, and Dr. S. A. Levine of Boston.

Adjournment.

CONGRESS OF AMERICAN PHYSICIANS AND SURGEONS.

Report of Treasurer, January 1, 1929.

RECEIPTS.

Cash balance from previous treasurer	$ 149.00	
Assessments of constituent associations of the Congress	1,055.00	
Registrations	4,485.80	
Interest on bank deposit	240.45	
Total Receipts		$5,930.25

DISBURSEMENTS.

Tuttle, Morehouse & Taylor (reprints, etc.)	30.80	
Rubber stamp	.90	
Postage stamps	2.50	
Paid by Dr. A. R. Shands:		
Printer's bill	257.00	
Printer's bill	25.25	
Dr. Shand's secretary's salary	150.00	
Clerks, Bureau of Registration	70.00	
Postage	52.00	
Use of lantern	40.00	
Incidentals: registration book, rubber stamp; dup. checks, etc.	10.00	
M. G. Thompson, clerical work for treasurer	15.00	
V. B. Alvarez, Reporter of Congress	46.00	
Tuttle, Morehouse & Taylor; programs and envelopes	640.53	
Mayflower Hotel, electrician	19.56	
Refund of overpayment of registration to Dr. E. Rixford	.50	
Total disbursements	$1,360.04	
Balance on hand—on deposit in Savings Dept. of The First National Bank, New Haven	4,570.21	
		$5,930.25
Estimated cost of publication of Proceedings of Congress	$2,950.00	
Estimated balance after payment for publication of Proceedings of Congress	$1,620.21	

J. E. LANE, *Treasurer.*
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American Ophthalmological Society ..	185	..	1	106
American Otological Society	138	..	8	72
American Neurological Society	203	..	21	109
American Gynecological Society	100	..	26	54
American Dermatological Association..	94	17	6	61
American Laryngological Association..	118	22	2	84
American Surgical Association	217	..	21	145
American Climatological and Clinical Association	185	8	18	111
Association of American Physicians....	278	2	16	181
American Association of Genito-Urinary Surgeons	76	9	8	49
American Orthopedic Association	128	8	5	74
American Pediatric Society	97	3	10	62
American Association of Pathologists and Bacteriologists	402	..	..	..
American Society of Tropical Medicine	246	..	15	54
American Association for Thoracic Surgery	117	..	6	72
	2584	69	163	1234
Total membership of Congress		2816		
Guests and Visitors				121
Total attendance				1355

THE PRESIDENT'S ADDRESS.

THEOBALD SMITH, M.D.
Princeton, N. J.

THE DECLINE OF INFECTIOUS DISEASES IN ITS RELATION TO MODERN MEDICINE.

In endeavoring to fulfill the highly honorable task of addressing you I have queried whether it is humanly possible for any one man to profitably entertain even for a half hour such a gathering as this whose training and information extends to the remotest corners of medical science and practice. The answer to my query was that what you expected was something, however trite and commonplace, that was stamped with the experience and conviction of the speaker, something which he had lived through and perhaps helped to vitalize with a contribution of his own. It was with this answer before me that this address was written.

I.

In taking as my theme the subject of infectious diseases I realize that it does not play on the medical stage the part it did a generation ago. Since then there has grown up a most important body of knowledge in the pathology of the individual rather than of the mass. Genetics, endocrinology, nutrition and hypersensitiveness, to mention only the more important, vie in attractiveness of field with the more or less forbidding and forbidden territory of communicable diseases. Nevertheless we must accept the fact that although the microbic aspect of disease may appear to have spent some of its force in research, the organisms are still with us and cast their myriad shadows over every department and specialty of medical practice while an expensive machinery of community, state and nation is permanently set up and in operation to cope with them.

Standing as we do near the end of half a century of work in scientific microbiology we may well pause to consider the stupendous spread and depth of accurate information which stands to the credit of the many workers since the triumvirate Pasteur, Lister and Koch began their pioneer work. About fifty years ago Lister's method of anti-

septic surgery had already penetrated through western Europe. In 1880 Pasteur began his work on chicken cholera and anthrax vaccination. In 1876 Koch had demonstrated the cycle of the anthrax bacillus from spore to spore in a drop of aqueous humor under a coverglass. In 1881 he first described the use of solid culture media, such as potato, and introduced the use of gelatin. These enterprises represent the total of precise knowledge concerning the causes of infectious disease at that time. In the early eighties the medical student was just beginning to hear of the "germ theory of disease." There is no occasion before an assembly like this to review the progress of our knowledge since this early period. I need only mention the isolation and culture of many pathogenic microorganisms, the study of acquired resistance to disease and the antibodies associated with it, of anaphylaxis and hypersensitiveness, the rise of chemotherapy and the development of our knowledge of the protozoan and ultra-microscopic world of infectious agents.

Giving birth to the concrete achievements are the concepts, theories and hypotheses, which in part true, in part false, animated the workers and supplied the energy which was to bring forth the next crop of new and frequently unlooked for facts. These theories, held and defended by some master mind, formed the nucleus of so-called schools. The schools persisted because the theories for the time being were beyond demonstration. To-day we hear little of schools because even the most esoteric doctrine is very soon dissected by new and adequate technique and the school tends to dissolve before it has time to gather adherents.

It may be well on this occasion to recall some of the guiding theories which have survived and crystallized into actual facts and principles, for they form the foundation on which my subject rests. In the early days of the new era infectious diseases were looked upon as straight contests between host and infectious agent. The microorganisms entered, multiplied, and were checked and killed, or else the host succumbed. Now we know that in many diseases a kind of equilibrium establishes itself in which the microorganism remains somewhere in the host and is ready to multiply again when the host is off guard. In other diseases localizations occur in which the specific bacteria multiply and survive for a time. The victims of these conditions we now call carriers. The concept of carrier was developed chiefly with the protozoan diseases. In the bacterial diseases it first took root when the intensive study of diphtheria was

forced by the discoveries of toxin and antitoxin. The so-called focal infections are only types of carriers, for they represent local forms of disease towards which the host is partly immunized.

Coupled with this clinically serious problem of continued localized infectious processes is the one once actively debated whether the tissues of the presumably normal body are sterile. Elaborate experiments were made to test this question. As usual when the technique is not secure from criticism the interpretation of results follows individual leanings. So in this controversy. We now know that various bacteria, notably spores of anerobes, may maintain themselves for a time in the system without multiplying. They may enter through fresh wounds of the skin and mucous membranes. The liver, draining the digestive tract through the portal system, more often contains organisms of intestinal origin. The bacteria are always very scarce when present at all. They, however, serve to explain certain rare complications following surgical operations and urinary infections in the absence of other manifest causes.

The concept of carrier is in itself complex, involving as many possibilities as the microorganisms capable of surviving in the tissues. There are certain hardy species which remain within the system, others only on or in the folds and glandular structures of mucous membranes. Our civilization is continuously creating new *loci minores* in the body and no one can foretell what modifications now well-known pathological conditions may undergo.

There is perhaps no one subject in the domain of pathology of such importance as the problems involved in the defense developed and maintained by the body against invasion of foreign living matter. Its importance has been recognized from the very beginnings of the bacteriological era, since no field has been more industriously tilled experimentally than what is now called immunology. Before any headway had been made in the analysis of disease processes associated with infection numerous attempts were under way to protect the body against them. The remarkable success of Jenner's cowpox vaccine long before any organized, substantial investigation of infectious diseases had been started, stimulated Pasteur to undertake studies in fowl cholera and anthrax. Since that time there has been no end to the procession of methods for the protective inoculation against disease. Here as in other fields empiricism was far in advance of organized scientific inquiry and harvested some notable triumphs of permanent value.

It was in 1884 that Metchnikoff published his first paper on phagocytosis. In a disease of a small crustacean occurring in an aquarium he saw the spores of the microbe as they penetrated into the body surrounded and engulfed by wandering cells. This new theory of cellular defense was highly intriguing and kept all working in infectious diseases busy unearthing new data to support or controvert it. The discovery of antitoxin as a purely humoral agent of defense in 1890 added to the interest in that the controversy now waged around the relative significance of these two modes of action. Since then we have learned that the processes started by invasive organisms in the body are a series of highly complicated biological reactions, no one of which can be generally credited with being protective or offensive. Thus phagocytic cells may take up bacteria in large numbers regularly yet the process prove invariably fatal. Protozoa make a specialty of living within cells, both epithelial and wandering, during a portion of their cycle within the host. The phylogenetic significance of the group of mobile and mobilizable cell types was referred by Metchnikoff to ancestral processes of nutrition in very low forms. Subsequent specialization towards defense in the animal series was scarcely capable of meeting successfully the many diverse invasive organisms and the defending cells actually became in some instances a support and comfort to the enemy. On the other hand the theory of humoral defense encountered an equal amount of contradictory evidence concerning the bactericidal activities of the blood. We are thus constrained to acknowledge that while we know a large number of interesting cellular and humoral actions and reactions, we have not yet learned to piece them together for any one disease satisfactorily. The task therefore is still before us to study each host-parasite relation in detail and avoid generalizations, except as stimulating hypotheses, until the different types of reactions have been studied comparatively.

Aided by studies in genetics we are beginning to understand that we cannot add to the original dower of capacities we received from our ancestors. Medicine may assist greatly by determining the maximum capacity of these innate powers and furthering them by adequate stimuli. It cannot add to the capacity to produce protective bodies through vaccination but it is in position to stimulate and mobilize primary latent protective energies. Fortunately individuals of the same species do not differ among themselves to such a degree as to jeopardize current methods of vaccination, but it must always

be borne in mind that towards any infectious disease a few individuals may be inadequately provided with a capacity to protect themselves even under maximum stimulation by vaccination procedures.

It is still a question whether the immunity or susceptibility of individuals is correlated with any anatomical or physiological character. In general the impression acquired by experimentation is that relative susceptibility is a character by itself and not predictable from other bodily functions. It has been customary to speak of natural as distinguished from acquired immunity. Certain species are definitely immune to diseases to which other species succumb. This type of immunity is probably dependent on quite different factors from that possessed by resistant individuals of a susceptible race. In the latter the immunity evidently depends on the capacity of the individual to reproduce and mobilize certain protective antibodies quantitatively. In the naturally immune species these are not needed. Metabolic processes probably control the situation. Man and all animals have a certain fundamental resistance even when the race has not been in prolonged contact with the specific microorganism since dosage of virus makes a great difference in the outcome of all inoculation experiments. What is really meant by the term acquired immunity is that the individual has simply strengthened or augmented his original resistance in the presence of specific infection. This changed state is usually indicated by an increase in certain antibodies.

As a corollary of the various established laws in immunology it has been generally accepted that the animal organism is a unity in its efforts to protect any tissue or organ. The general effect of Jenner's local vaccine pustule must have forced this principle forward long ago, but with the splitting up of the practice of medicine through necessity into specialties dealing with individual organs this fundamental law may be easily lost sight of. The development of immunology has however accentuated the fact over and over that any local multiplication of infectious agents tends to spread its influence through the entire system. A single small tubercle allergizes the whole body. Antibodies are poured into the blood so quickly that the higher local immunity produced by inoculation can be distinguished only with great difficulty, if at all, from that pervading the organism as a whole.

Next in importance to the concept of immunity is that of the variability of microorganisms. The period before the eighties of the

last century witnessed the publication of what we would to-day designate as very wild theories on the changes which microorganisms may undergo. One of the earliest writings of Koch is directed against a book of Nägeli's in which among other things it is stated that the anthrax bacillus becomes a saprophyte in putrefying animal tissues. To counteract these heterodox assumptions Koch and his followers became rather too rigid adherents of the theory of the stability of pathogenic bacteria. It required some time therefore for the newer ideas of variability to get a foothold. Slowly material accumulated which proved that no pathogenic microorganism was without variants. The source and origin of the many varieties of the same species remained obscure. We know that bacteria gradually lose their virulence or mutate into almost non-virulent types under conditions of artificial culture but this decline is to be expected. This is different from the variations observed among microorganisms producing the same clinical disease and not yet influenced by artificial culture. The variations among pathogenic microorganisms are probably induced in the human and animal body. Meeting in the tissues a variety of obstacles, the parasite must adapt itself or else cease multiplying. It is highly probable that serological differences of microorganisms otherwise closely related biologically are the result of interaction between them and the host tissues. This capacity to undergo modification will be touched upon again.

The variability of microorganisms meeting a similar variability in the natural and specifically augmented resistance of the host produces a certain relativity which occasionally defies all our standards. The intimate association of the newborn generation with its immediate ancestors naturally favors the direct transfer of infectious agents, chiefly mucous membranes parasites carried usually with light symptoms or none at all by the adults. Nature has provided a partial safeguard in the placental transmission of antibodies to produce a temporary passive resistance. In those species which have placentas impervious to the protective antibodies, the colostrum transmits them. In experiments carried on for some years on one of this latter group, the remarkable protection afforded calves by the ingestion of colostrum has been demonstrated by replacing it with ordinary milk. Under such conditions *Bacillus coli* is as virulent as the anthrax bacillus in later years and produces a fatal septicemia. In the partially protected a latent microbism establishes itself and it may manifest itself later up to the third month of life in multiple arthritis,

nephritis, pneumonia, and possibly other affections. The door to these invaders is probably closed as soon as peptic digestion is well established. Before that colostrum fills the gap. The placental mode of protection, fortified perhaps by colostrum, must have played an important rôle among primitive people in safeguarding the very young against endemic infections, at least against a fatal outcome of the same. These results show that bacteria harmless later on may seriously cripple and even destroy the young. They also suggest that *Bacillus coli* may at some early period have produced a cholera similar to that associated with the cholera vibrio to-day.

As a result of the impact of the new concepts derived from microbiology the medical mind turned to a large degree from the idea of cure to that of prevention. First, to check infection by stopping the transfer of microorganisms from sick to well, disinfection dominated preventive medicine for many years. This phase led to a great development of public health agencies and the entire public health service with its water supply and sewerage systems and its multitudinous inspection of food products. The idea of prevention came thus with full force into medical practice. The physician expects not so much to cure as to prevent the disease from taking the next downward step, to prevent distant localizations by keeping the virus in its original attacking place until developing immunity takes a hand. The future then even of the next day or the next hour became the object of inquiry so that it might be adequately met and the untoward processes checked. Prevention implies the study of causes or necessary antecedent conditions not only between health and disease, but between each stage of the disease itself and the next, hence the modern craving for more information concerning the earliest stages. We know very little, if anything, of what transpires in the incubation period, so definitely fenced off from the first clinical manifestations. Yet it is conceivable that in this stage the fate of the patient if left to himself may be diagnosed by refined studies. Unfortunately the difficulties are great. To trace the entry and local behavior of a few microbes at the start is a far more trying undertaking than to follow the exit of myriads in the recovering host. The presence of minute amounts of protective substances or of toxins in the early stages is probably beyond the detective capacity of present laboratory methods.

Two types of procedure in the scientific inquiry into disease processes have definitely come out of the idea of prevention. First,

inquiry into the causes and antecedents of disease upon which is based the public health movement to eliminate them. Second, empiric discovery, largely by trial and error methods, of the means to neutralize the effects of causes, such as vaccination and chemotherapy. These two movements are intimately associated, so much so that we hardly know which one we are trying to further in research, not infrequently both at the same time. Springing from them are two currents governed by distinct attitudes of mind: The first endeavors to bring the race back towards earlier, more primitive and more fundamental modes of life under which its evolution took place. The second tends to develop substitutes for these and to permit us to involve ourselves more completely in our artificial enviroment.

During the half century of microbiological dominance the rise of special workshops or laboratories has been phenomenal. It would be difficult to exaggerate the part played by the pure culture of bacteria upon the establishment of laboratories. It furnished readily to a large number of workers a single isolated living factor with which to experiment. Contrasted with the complicated technique and equipment of the physiologist, that of the bacteriologist was simple and readily reproduced almost anywhere. The same is true of the immunological laboratory, the special outgrowth of microbiological research. In evaluating the functions of the research laboratory we must bear in mind its limitations. It usually deals with more or less exaggerated, intensified types of induced disease, whereas in medical practice much material represents the terminal or hold-over stages of infectious processes, those most difficult to deal with therapeutically. The laboratory deals with minimal fractions of the blood, for example, whereas the body utilizes the entire blood through rapid circulation. Resistance is thus frequently demonstrable clinically when tests of minute samples of blood fail to reveal this resistance because of the apparent absence of antibodies. In fact these antibodies can be brought within the range of laboratory methods only by procedures of hyperimmunization.

The laboratory functions in two ways. It undertakes to analyze the complex phenomena of nature as perceived by the senses with the aid of methods and instruments of precision. It thus may depart from the problem as a whole, in proportion to the refinement of its technique. In fact, through artificial means it simply gathers individual facts to aid it in the final study of the natural phenomenon. Its second function is to imitate the complex natural phenomena as

closely as possible. In this respect it is like the concept of limits of mathematics, we may approach the actual more and more as our technique develops without however actually reaching it. Both these considerations must be borne in mind in applying results of laboratory experimentation to everyday problems in medicine. Only by close coöperation between the laboratory worker and the clinician can the more precise laboratory methods be made useful. This coöperation usually results in the domination of one or the other of the agents whereas the relation should be a pure partnership.

II.

It is obvious that our knowledge of the plasticity of microorganisms, of their survival in the recovering host, or even in the normal host, and of the limitations of the body in raising its resistance is of essential significance in a study of the decline of infectious diseases. This decline involves more than the word stands for. It means the suppression of new as well as the blocking of the return of old, well-known diseases. Protagonists of the part played by medical science and practice in the decline of infectious diseases have in general failed to make use of their strongest argument, namely, that it has probably suppressed many diseases *in statu nascendi* and that the falling curve of certain endemic diseases is only a surface phenomenon of what is going on underneath. It is this latter aspect of the subject which I wish to discuss.

If we study the entry of a new disease into a population of animals the first phenomenon is a rather high mortality. After a variable period this simmers down to sporadic cases here and there, to modified types of the disease, and finally to complete disappearance or else rare casualties. Each disease takes a slightly different downward course. The upshot is a lowered mortality, and an increased morbidity if we include in this latter term all localizations and carriers. The course of a human epidemic or pandemic is much the same and subject to many variations depending on the nature of the microbic cause. The cause of the decline of the epidemic form has been referred to the gradual rise of specific resistance of the host as a result of mild infections. It has also been claimed that there are certain more highly resistant individuals who, being spared, transmit this resistance to their offspring and so build up an immune population. Certain it is that populations exposed through migrations or intercourse to a disease strange to them suffer in epidemic form. The population fur-

nishing the infection may not even be outwardly diseased. This local or geographical immunity is especially marked in the protozoan diseases, although it exists towards all infectious diseases.

As to the great epidemics which tend to sweep rapidly over large areas of the earth's surface, I believe that the causal organisms have a special history. They have come from an environment different from that into which they are launched when they produce widespread disease. Leaving them for the moment, we may call attention to the general law of nature that too virulent races of microorganisms may become extinct because the host perishes before the period of excretion begins. Other microbic races then take their place. These are probably the result of the interaction between host cells and fluids and the microorganism by which virulence is slightly reduced. The end result is thus not only a slight rise in the specific resistance of the population but an adaptation of the microbic cause to living conditions in the new host which means virulence lowered to a certain equilibrium level. After this has been reached, provided the microorganism is capable of the required modification, the disease becomes endemic with small endemic outbursts from time to time. It is this form of disease in which gradual reduction in mortality has taken place and in which economic and medical influences play a part. The larger pandemics are in a measure checked by quarantine and isolation but the progress of the infectious agent is usually so rapid that the barriers are put up after the entry of the virus and the epidemic goes its way more or less unchecked. These facts are well known. The one addition I suggest is the gradual or more accentuated modification of the epidemic virus into a more tractable, less destructive organism, probably never to return to its original level of virulence. Viewed from this angle, the flora of the mucous membranes may be either the degenerated survivors of recent or ancient epidemic types or else saprophytes adapting themselves to the parasitic state. As already hinted at, even the ubiquitous *Bacillus coli* may have a history.

The infectious diseases of the human race, some probably originating in its animal ancestors and passed on to primitive man, developed in an environment equivalent to that of animal life. Transmission of infection by contact and otherwise was immediate, direct, without distant circumlocutions through milk, drinking water or stored foods.

The virus was fresh and uninfluenced by prolonged exposure to environmental agencies. There was, however, a significant difference between the primitive and the civilized groups. The former were

essentially static; the modern primarily mobile, even earth-encircling. In the early history of man and his ancestors, different infectious agents probably arose in different parts of the globe, much as a distinct fauna and flora characterized the various continents and smaller isolated areas. They remained more or less confined on account of the limited capacity of man for going long distances. Even migrations and wars were slow in progressing and diseased individuals may have been left behind or they may have succumbed on the way. The discovery and use of any means of rapid transportation might have been highly destructive or even fatal to the early races of mankind. Another difference to be noted is that early man lived in close contact with his domesticated animals and with many untamed species both of vermin and game quality. Even to-day among people of the Far East the domestic animals are housed under the living quarters.

In view of these facts the question arises, what are the possibilities for the appearance of new or modified types of infectious disease when microorganisms transferred to another species change their physiological character more or less and accidentally become through this change, infectious for other species, including man? That this process of host changes is responsible for the many microorganisms, closely related to one another yet not absolutely identical, seems to me highly plausible. We are familiar with three races of tubercle bacilli. They are probably derived from some one ancestral type. To what extent has each one reached the limit of its capacity to become modified in another host? Or is it still possible that under special conditions, realizable only under a very unusual combination of events, the bovine type might become human? Experiments have thus far negatived this but experiments are limited in combinations and time whereas in nature the number of opportunities and the time are unlimited.

While many factors working in primitive times to bring animal infections within the daily reach of man are now eliminated others have taken their place, such as the extreme mobility of modern life bringing together elements from the entire globe. This is evidenced by the rapid introduction of insect pests and plant diseases. There are even now a number of animal diseases capable of producing serious disease in man, but there is in all something lacking, except in pneumonic plague, to complete the cycle in man and make it a human disease. The animal still acts as the reservoir of the infectious agent.

The well-known pandemic types of disease, such as Asiatic cholera and influenza, which have in the past repeatedly swept over Europe and reached the American continent, come from regions where primitive conditions are the rule. Here the organism with pandemic possibilities may receive its preliminary training in animal species and eventually reach man. That it always exists and is kept confined only by quarantine is the usual explanation. The history of animal plagues hardly warrants this as entirely satisfactory, for the great pandemics appear to have a potentiality which defies quarantines and vaccines.

A good illustration of my theme is the recent, more or less sudden, sporadic appearance of undulant or Malta fever at great distances from its supposedly primary center about the Mediterranean. Instances of this clinical complex have been reported within recent years from various regions in the United States, from Denmark, Germany, and Rhodesia. The number of cases in Italy and other endemic regions appears to have increased. How are we to account for these occurrences? The organism of Malta fever in goats has been recognized since 1889. The closely related form producing abortion in cattle since 1897. Another race in swine was first recognized in 1914. It is my belief that the porcine variety has been developed from cattle in recent times in the Middle West as a result of the very close association of the two species in the feed lots and the adaptation of the bovine variety to the pig under certain unknown conditions. If the bovine type is responsible for undulant fever in man, why is it that such cases have been brought to light within three or four years, whereas according to evidence developed in my own work this microorganism was active in dairy herds in this country as far back as 1893? Since then probably every one who has drunk raw milk has ingested this microorganism once or many times. Is it that the hospital laboratory has completely overlooked it during these 35 years and has medical practice so many erroneous diagnoses to answer for? My own tentative explanation is that the recent cases come from several sources. It is probable that the bovine organism is only feebly, if at all, capable of multiplying in the human system, that passages of this race through the pig have made it more virulent for man and that some of the modified porcine races in one way or another have gotten back into the udder of some cows establishing small centers of human disease. It may

also be that the caprine or true Malta fever variety has been spread about in ways I need not go into here.

If nothing had been known of the animal diseases furnishing this human infection what would have been our speculations on this new disease? We should have regarded it as non-infectious since it does not pass to contacts. We might have inferred that most human beings are immune and that some are potential carriers. We should probably have failed to turn to animals as reservoirs owing to the absence of any direct clues. If the virus had been filterable, non-cultivable, the darkness would have been intense and persistent.

Concerning the disease in man several questions at once present themselves. Does *Bacillus abortus* possess the capacity to penetrate into the system but without being able to multiply until some conditions arise depressing or perhaps exalting certain functions which permit the microorganism to multiply and call forth symptoms of undulant fever? It is obvious that a few bacteria in the normal system would only by accident appear or be sought for in cultures of blood. Antibody production would naturally wait on multiplication. Bearing in mind the fact that the bovine race of *Bacillus abortus* is known to have been in cows' milk at least forty years, there is still another explanation which deserves consideration. If the appearance of undulant fever should prove to be recent and if it can be traced in some of the cases to the bovine type of *Bacillus abortus* in cows' milk, there may have been a decline in the natural resistance of the race, a decline entirely within the bounds of probability on account of the changing mode of life and the gradual elimination of natural selection with reference to infectious diseases.

Taking these facts into consideration and the increasing number of new trials open to microbes to become established in new hosts made possible by the continuous flux of an increasingly dense population of both human and animal species, domesticated or merely parasitic on conditions produced by domestication, we may begin to visualize the possibilities for new diseases to appear. They might come more or less suddenly when conditions are rapidly altered as in war for man, and in rearing large numbers of animals in confinement with regular and frequent communication with other similar populations. We may readily conceive of the many changes in infectious organisms partly accomplished, followed by the destruction of the new race because some conditions are not fulfilled, and hence the many failures which precede what we may call the birth of a new disease.

In this highly significant process the original character of the aberrant parasite may be so altered that its source cannot be traced. Its course may have been devious and the intermediate stages unobserved when they occurred and since wiped out, so that this course can no longer be traced back to its beginnings. The appearance of apparently new diseases associated with ultramicroscopic organisms is particularly mysterious, since our methods are quite inadequate to inform us from what direction the newcomer hailed. The ancestry may be directly before us, in our midst, in fact in some animal disease, but we may fail to see it because of the irreversible process that has brought the change about. With this concept in mind we can also imagine many dangerous combinations frustrated, cut short, by hygiene and sanitation, numerous diseases in gestation prematurely aborted, but occasionally one successfully escaping destruction and launched into the world, at first unrecognized until a certain momentum has been acquired which brings the disease into the open.

III.

We are now in position to view the difficulties to be encountered in discussing the factors that have led to a gradual decline in the mortality due to infectious diseases. This decline we may readily grant without entering the statistical field. It comprises all those endemic infectious diseases to which the population has become partly immunized and which occur from time to time in limited epidemics. Three agencies are cited as claiming more or less of a victory: changes in economic conditions, the applications of medical science, and the interplay of natural forces still largely unknown and not controlled by human foresight which tend to raise resistance of the host and reduce virulence of the parasite. Improvements in economic conditions provide opportunity for a wider, aseptic zone about individuals through personal cleanliness. On the other hand, industrial changes lead to huge concentrations of the population in which the individual aseptic zone is broken into at many points. One result is a continuation and probable increase in respiratory infections. Decrease of intestinal infections has been accomplished through adequate water supply and food inspection. The sewage problem is still unsolved and all we have done is to convert our water courses into open sewers with occasional explosive outbreaks of intestinal disease as the result. The time is coming when the intimate relation

between water supply and sewage disposal will suddenly develop acute crises in the sanitary affairs of large communities.

The widening aseptic zone I have described tends to delay the passage of infectious agents from individual to individual. The influence exerted by various agencies on infection when it has to travel the longer distance from individual to individual, as for example when the typhoid bacillus comes back by way of river water receiving sewage, instead of being conveyed directly, is a problem difficult to attack because methods for measuring degrees of virulence must be carried out in alien species all of which are naturally insusceptible. The same difficulty arises when we wish to measure the virulence of microorganisms carried by recovered or healthy individuals. The natural inference, it seems to me, is that there is a gradual attenuation going on which is proportional to the period since convalescence and that the most virulent types are the ones transmitted during or immediately at the end of the acute disease process. Diphtheria bacilli have been found atoxic from throats a variable period after the acute process has subsided.

Civilization from the medical aspect may be defined as the maintenance of an increasingly dense population with a falling death rate in spite of free intercourse. Taking this definition as a base, we are safe in saying that without the steady development of medical science and practice civilization would have been unable to move forward. In every detail of individual and communal life medical science has formulated protective devices to maintain health largely by the suppression of infection. Without the constant application of medical and preventive safeguards the human race could not sustain itself. If it should drop to the level of animals in this respect and throw safeguards to the winds its fate would be reduction to animal density in population or even worse unless the race segregated itself into non-communicating groups and each one allowed the existing viruses to burn themselves out as it were. The number of diseases scattered over the globe is so great that free intercommunication on the animal level might bring so many to bear on the race as to make impossible its struggle against other natural injurious agencies.

The bringing together of large numbers of individuals from widely different regions, as in war, is perhaps the most fertile method of resurrecting old local diseases, intensifying prevailing types and even creating new ones. The conditions during war also favor a broader contact with animals both useful and noxious. Breeders of livestock

have learned to separate large flocks whose ancestors were gathered from various regions to improve the breeds into small colonies to escape the unavoidable, almost catastrophic epidemics among large numbers.

Medical practice performs another function which is apt to be overlooked by the laity and not infrequently antagonized by it. I refer to the universal custom in private, hospital and sanatorium practice to isolate the sick in such a way as to exterminate in many instances the infectious agent of the patient. How much is gained by this procedure no one can tell, but many an organism of superior virulence is obviously stopped in its course and destroyed and not only other individuals but possibly large groups protected from epidemic invasion. Where the microorganism represents a resistant type, as in tuberculosis, and the chronic carriers are the rule, the advantages of isolation and destruction of the secretions carrying the virus consist in a reduction of the general level of infection that to all appearances accounts for the large number of allergic individuals free from demonstrable lesions. This spontaneous vaccination in countries where the disease has prevailed over long periods is probably a safeguard rather than a danger. With the discharge of large numbers of bacilli in open ambulatory cases, freely expectorating as was the habit fifty years ago, the danger of large dosage and consequent clinical progressive disease is obvious.

It is difficult from any point of view to establish the claim that parasites are beneficial or even harmless to the host. Although parasitism is a fundamental phenomenon of life, this is no argument that it is beneficial to the host species. Nature ignores man as such. He is simply an animal and therefore subject to laws governing parasitism. He is however the only species more or less successfully struggling to eliminate parasitism in all its forms from his body. It is probably the apparent hopelessness of the effort that has led some scientists to state that it might be wiser to cope with infectious diseases in the animal way by the elimination of the weaker hosts and the increased resistance of the survivors than to try to exterminate the parasites. The defenders of this view have failed to recognize the fact that the number of infectious and parasitic agents in existence have never become widely diffused either among men or our domestic animals. The *laissez-faire* method would bring into play many hitherto exotic diseases in addition to the endemic types. Moreover, even if through natural processes virulence of any epi-

demic type of microorganism tends towards a lower level, we should still have the modified, even if non-fatal, forms of disease to contend with, including localized disease and focal infections, all of which are serious handicaps in the struggle for existence. Just what forms of human society would have been the outcome of the progressive economic development with its speedy, unrestricted intercourse and no restrictions on the activities of infectious agents, no one can visualize. It is my opinion that the population would have become stationary or retrogressive and economic development equally so.

In short, medical practice in all its forms is so thoroughly a part of our civilization that no surgical operation could remove it. Attempts would probably endanger the life of the social organism as at present constituted. Medical science and practice must go on and continue their evolution parallel with human society, for we are perpetually mortgaged to maintain the barriers against our environmental enemies which will be continually thrust on us, until a kind of world organization shall have recognized as important factors even the most backward races as potential dangers and shall have brought the entire world population to a common anti-parasitic level.

It will be inferred from what has been said that we have still much to learn concerning the world of parasitic microorganisms, their rise, decline and disappearance, the modifications they undergo and the changes in the character of the diseases involved in this flux. It is not an immediately helpful study in the treatment of disease, but it represents the foundation of medical science as applied to infectious diseases. It is the natural history of these diseases which we lack. In the natural sciences, the foundation was laid by studies of a broad inclusive comparative character into the existing fauna and flora, which gave us among other things the theory of evolution. Later this was followed by more exact, analytical studies in the laboratory. In medicine the process has been the reverse,—cure, treatment, vaccination and prevention, and lastly research into the nature of the disease. It is true that medical practice is the study and care of the individual, but medical science is rather a study of the race and therefore a study primarily of disease itself. The race can only be studied through its individuals but science must focus first of all on the disease-process rather than on the immediate relief of the individual. Medical science, as one of its tasks, needs to study disease comparatively as well as experimentally and not limit itself to one species as this is a barren process beyond a certain stage. In

fact the progress of medical science to-day depends almost wholly on animal life. To materialize this plan in the study of infectious and parasitic diseases it will be necessary to exploit all spontaneous animal diseases, to study the disease of the individual in all its forms, the effect on the population as a whole, the rise, decline and modifications of the virus, and eventually a comparative study of similar diseases in different species. It will be highly desirable to investigate the diseases of untouched animal populations which are closely associated with man in more or less primitive conditions, for our own domesticated species, both useful and noxious, are under the influence of our civilization which makes research almost as difficult as that of humans, but for very different reasons.

It is obvious that the research laboratory must play a large part in such a natural history study of disease, but it must subordinate its analytic tendencies to the broader view of the whole and assist in the comparative study. Its results will then serve as problems to be further analyzed by a more refined technique. It is needless to say that this plan may be classed as a luxury rather than a necessity in medical research. Those of us who have followed the development of research over a considerable period of years have frequently been faced by the fact that what we regarded as the luxury of one period became a necessity of the next. Although much has been done in the direction indicated by pioneers, risking their health and lives in the wilds of uncivilized continents, the work perforce has been fragmentary and desultory. I think that coming generations will see established research stations in hitherto only partially explored territories where living conditions will be created to make research not only possible, but remunerative to medical science, and where all forms of disease in various species will be objects of research.

THE HEART.

ECONOMIC ASPECTS OF HEART DISEASE.

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New York.

No small part of the present colossal wealth of our country is due to the accumulated saving of the lives of men and women in the years of their greatest material productivity, by the fall in the death rate from pulmonary tuberculosis. It is unlikely that in this country and in our time any other change in the prevalence of disease can produce such impressive increase in the earnings and savings of our people. Now that tuberculosis has lost its leadership of the column of deaths, and heart diseases have emerged, partly by the fall in the death rates from other causes, and partly because of the great and sustained increase in the death rates from heart disorders as the most frequent of the causes of death, popular interest, as well as medical, drives us to analyze causes and costs in this field of pathology as a community problem.

In 1921 it was estimated that the per capita cost of tuberculosis to the people of the United States was $7.96 annually, and that this burden, if distributed among those persons with an active, clinically recognizable tuberculosis, amounted to $1,262 annually per patient, and that the total loss due to tuberculosis during the entire life span of the population at the then rate of tuberculosis mortality would amount to $27,125,000,000.*

No better confirmation of the adage "The first wealth is health" is to be found than the estimate of economists that the value of human lives in this country is at least five times that of all material possessions.†

While the implications of the title of this paper may be as broad as the range of human activities, it will be understood for present purposes that "economics, or political economy is the social science which treats of that portion of human activity which is concerned

* Emerson, H. A Brief for Investment in Adequate Prevention of Tuberculosis. Am. Rev. of Tub., Vol. VI, No. 6, Aug., 1922.

† Dublin, L. I. Value of a Man. Metrop. Life Ins. Co. Stat. Bull., Vol. VII, Nos. 6, 8, June and Aug., 1926.

with earning a living," and that under the term heart disease are included those various conditions provided for under the four titles of the International List of Causes of Death, 87-90, Pericarditis, Endo- and Myocarditis (acute), Angina Pectoris and Other Diseases of the Heart.

We are necessarily limited to the use of these few terms, inadequate as they are, because with rare exceptions under no others are there to be found records of the distribution and duration of heart diseases which permit us to estimate the character and the extent of the influence of this particular group of disabilities upon earning capacity and span of life.

The bearing of disease upon the earning of a livelihood depends at least upon the age of an individual at onset, the degree and duration of disability as the process develops, and the extent to which death from this cause shortens life.

Information is therefore to be sought from the records of deaths, and of sickness, where these latter are assembled through institutions or agencies such as hospitals, dispensaries, visiting nurse associations, industrial and insurance or compensation offices.

While no economist or statistician can express in figures alone the value of man's life, there is a trustworthy method of calculating in terms of earnings, the relative returns on the investment of life when death comes to man at different ages.

The studies of Dublin and Lotka* will be used as the basis of estimating the economic losses due to heart disease in this paper. By these authors the money value of a life at a given age is considered to be the difference between the person's calculated future earnings at the time of his death, and the present worth of his probable future expenditures.

Since the overwhelming majority of men in the United States to-day are found in, or at least nearer the $2,500 income class of earnings than in the $5,000 a year class or over, we shall confine our estimates to the former without attempting to make allowance for that relatively very small proportion of heart patients and deaths occurring in the higher income group. For convenience of reference, however, the following abbreviated table from the studies of Dublin and Lotka will indicate the relative values of these two groups of earners expressed in dollars of value at the age of death.

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* Dublin, L. I. Met. Life Ins. Co. Stat. Bull., June and Aug., 1926.

ESTIMATED VALUE OF A MAN'S LIFE IN TERMS OF NET FUTURE EARNINGS AT VARIOUS AGES.

Year of Age	On Basis of $5,000 Annual Earnings	On Basis of $2,500 Annual Earnings
18	$34,321	$28,654
21	39,176	30,818
30	48,562	31,038
40	45,670	25,795
50	30,354	17,510
60	10,256	8,499
70	5,988	562

Our knowledge of the degree of disability due to heart diseases during the various phases of their pre-clinical and recognized existence in man is too fragmentary to justify us in attempting calculations in this field. For persons completing the years of their normal life existence, i.e., with undiminished average length of life, though handicapped because of limitations in physical vigor, skill, or endurance because of disease, reliable estimates of money value at the various ages can be made as has been indicated by Dublin in his table on Substandard Wage Earners in Three Income Classes, $500, $1,000, and $1,500. (Statistical Bulletin, Met. Life Ins. Co., June, 1927, p. 2.)

The burden of heart diseases caused by the shortening of the life of occupied and retired civilians, 20-65 years (Report of Registrar General, England and Wales, 1921-23 on Occupational Mortality), is heavier among the unskilled workers than in any other social class, the unskilled workers' comparative mortality figure from this cause being 156.5 while that of the upper and middle class is 105.8 and that of skilled workers is 120.1, a difference to the disadvantage of the low income level group which is found also in tuberculosis, cancer and pneumonia.

This difference is particularly marked when the death rates from heart diseases by social classes are analyzed by age groups, the unskilled workers suffering out of proportion in all decades of life, but particularly in those under sixty-five years, as follows:

Age	Unskilled Workers	Skilled Workers	Upper and Middle Class Workers
16-19	25	18	6
20-24	34	25	15
25-34	55	37	18
35-44	91	61	43
45-54	181	127	112
55-64	454	394	401
65-69	946	907	977

As the Registrar General remarks, "It is evident that in early and middle life at least, heart disease as a whole is found to be especially fatal to the poorer classes, and although no record has been obtained of the social incidence of mortality from acute rheumatism, it is to be presumed that even if all classes were equally subjectable to this disease its effects upon the valves of the heart would be more serious to those least in a position to take the necessary precautions."

When the mortality from valvular disease of the heart is distributed by specific occupations the death rates from this condition are found to vary widely according to the character of employment.

If we consider the death rate from valvular heart disease of all occupied and retired males (20-65 years) as 1,000 we find the low mortality rates among

Insurance officials	230
Game keepers	249
Bank officials	334
Anglican clergy	391
Millers	393
Medical practitioners	438
Roman Catholic priests	472

Those occupations with a high ratio of mortality from this cause are:

Cotton Carders	2170
Cotton strippers and grinders	2063
Barmen	2024
Slate masons	1994
File cutters	1852
Cutlery grinders	1727
Slaters and tilers	1727
Cotton blowroom operatives	1688
Wool weavers	1666
Slate miners	1647

Similarly the occupations showing particularly high mortality rates from "Other Heart Disease" (chiefly myocardial) include many low paid and disadvantageous employments.

Tin and copper miners below ground	3244
Cutlery grinders	2834
Slate masons	2642
Barristers	2514
Hat formers and plankers	2317
Tin and copper miners	2242
China, kiln and ovenmen	2122
Pottery dippers, glazers, etc.	2108
Cellarmen	2029
Cotton carders	1869

From the less ample and conclusive evidence in this country we find as given in Pedley's* tables of proportionate mortality from principal diseases among ten occupations in New York City, 1924, that butchers, tailors, cigar makers, and shoemakers have death rates from heart diseases considerably higher than the rate for all the occupations listed as a group. Furthermore, it is seen from this same study that in 1920 the proportionate mortality for occupied males in the United States, ten years and over, was higher from heart diseases than for any of the other six leading causes of death.

The duration of heart disease as determined by various sickness surveys, reported by Frankel and Dublin (1915-17) in North Carolina, Pennsylvania, New York and Massachusetts,† differs radically from the duration of all sicknesses taken as a group. Calculations based on these surveys justify the following statements.

> One quarter of all sicknesses lasting less than two weeks; 3.7% of all heart disease lasting less than two weeks.
> 54.8% of all sicknesses lasting less than three months; 28.6% of heart disease lasting less than three months.
> 70% of all sicknesses lasting less than one year; 50.5% of heart disease lasting less than one year.
> 16.8% of all sicknesses lasting three years and over; 24.2% of heart disease lasting three years and over.

Among New York state factory workers in 1919,‡ 1.2% of total wages were lost on account of heart diseases, or $66.11 lost per case, while 2.2% were lost on account of tuberculosis, or $230.51 lost per case.

Sickness from organic heart disease among office employees in a large commercial office§ in New York City caused a loss of 253.9 working days per annum per 1,000 persons employed and showed a loss of 90.6 days per case of illness which constituted 2.68% of all working days lost, the differences between male and female workers being of no significance. There were lost on account of heart diseases 296.8 calendar days for each 1,000 persons employed, or 105.8

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* Pedley, Frank G. Trend of Occup. Mortality in the U. S., Jour. Ind. Hyg., Vol. IX, No. 11, Nov., 1927.

† Dublin, L. I., Frankel, Lee K. Sickness surveys under Metropolitan Life Ins. Co., 1916 and 1917.

‡ Sayre, H. D. Sickness Among N. Y. State Factory Workers in 1919. N. Y. State Dept. of Labor Spec. Bull., No. 100, Aug., 1921.

§ Personal Communication.

calendar days per case of heart disease, or 2.78% of all calendar days lost on account of sickness among these employees. There were 2.8 cases of heart disease per 1,000 persons employed in 1925 amounting to 3.4 cases per 1,000 clerk years or 0.13% of all cases of disability for that year. (Figures from Personal Communication in 1925.)

Analysis of health insurance claims on account of heart diseases* shows that 90.4 days are lost per claim, varying according to the age of the claimant from 42 days among those 25-29 years of age to 123 and 150 days among those 50-54 and 55-60 years of age respectively.

In Brundage's study of Sickness Among Industrial Employees† he found that according to the length of the maximum disability period provided for, the number of days of disability per case varied from 65 to 122 days, as compared with the duration of disability per case of 84 to 161 days in tuberculosis.

In the same report the average annual number of cases of heart disease as a cause of sickness disability per 1,000 male industrial workers (1922-24) was found to vary from 1.1 in the public utilities to 1.4 among the iron and steel workers, and 1.5 in other industries.

In Dublin's study of Causes of Death by Occupation‡ from the Metropolitan Life Insurance Company mortality experience, deaths from organic heart disease constituted 12% of deaths from all causes varying from 5.4% among railway enginemen and trainmen to 16.5% among farmers and farm laborers and for females 14.8%, ranging from 8.1% among bookkeepers and office assistants to 15.3% among housewives and housekeepers. These differences are doubtless due in large measure to the small number of cases dealt with as well as to the age groups of the persons involved.

A further analysis of Dublin's study reveals for all occupations the percentages of all deaths represented by those due to heart diseases, according to the sex and decade of life of the deceased as follows:

	Age Groups.					
	15-24	25-34	35-44	45-54	55-64	65 and over
Males	5.8%	5.4%	7.7%	11.1%	15.9%	20.4%
Females	6.9%	7.0%	10.0%	13.9%	18.7%	20.8%

* McManus, R. J. Proceedings of Casualty, Actuarial, and Statistical Association of America, 1919-20, pp. 177-196.

† United States Public Health Reports, 1926, Jan. 22.

‡ Bull. No. 207, U. S. Labor Statistics, Mar., 1917.

Similar information upon percentage of all deaths represented by deaths from heart disease and decade of death is found in Wynne and Guilfoy's* report for experience among employed males in New York City, 1914, as follows:

Age Groups.					
15-24	25-34	35-44	45-54	55-64	65 and over
8.4%	8.7%	10.3%	17.6%	22.2%	28.0%

In both these reports there are considerable differences according to sex and occupation but the variations are quite consistent among the age groups.

Regardless of age and sex, the variations in the percentage which heart disease deaths constitute of all deaths, by occupations, were found by Wynne and Guilfoy to be as follows:

All occupied persons (53,541)	17.9%
Blacksmiths	15.8
Cigarmakers and tobacco workers	18.9
Clerks and bookkeepers	14.0
Compositors and printers	13.6
Garment workers	20.7
Laborers	13.4
Machinists	14.1
Painters and paper hangers	14.9
Railroad track and yard workers	17.6
Saloonkeepers and bartenders	10.1
Teamsters and drivers	10.3

In Brundage's study of a ten-year experience with absences from work in the Edison Illuminating Company of Boston he found that there were for both men and women, four absences for heart diseases among each 1,000 on the payroll per annum, but that while the men lost 49.52 calendar days on account of each such sickness, the women lost 99.73 days.

Of the total deaths from all causes in the United States Registration Area, 1925, 1,219,019, there were 191,226 or 15.7% from heart disease. Of these (215 age unknown) 170,482 or 89.2% were in persons forty years of age or over.

40-49 years of age	16,384
50-59	28,118
69-69	44,843
70-79	49,774
80-89	27,262
90 and over	3,886
Unknown	215

* U. S. P. H. reprint, No. 400, June 8, 1917.

There were only 20,744 deaths from heart diseases in persons under forty years of age.

Under 10 years of age	3133
10-19	4095
20-29	4879
30-39	8637
	20,744

The decade 70-79 years, in which the largest number of deaths occurred, is the last one for which Dublin estimates that there is any balance of money value in favor of the individual between his probable earnings and expenditures.

The largest number of deaths from nephritis and from cerebral hemorrhage also occur in the decade of life 70-79, while the largest number of deaths from cancer occur in the decade 60-69 and of tuberculosis in the decade 20-29.

Not alone the total number of deaths from a particular disease but their distribution among the decades of life and the duration of the disease as a cause of relative or complete disability are the determining factors of its economic cost to the individual and the community.

In dealing with heart disease we must consider duration and death, moreover, on the basis of the three dominant etiologic groups, rheumatic, syphilitic and arteriosclerotic or senescent.

In translating clinical histories into graphic form for the purpose of expressing the spread of disability and death across the years of life, we have the following pictures of the three groups mentioned. (Chart Course of Heart Diseases.*)

When rheumatism completes its cycle from onset through a period of disabling symptoms to and through the time of decompensation to death, all under the age of 40, which occurs in the great majority of cases, we find that the onset is under 10 years of age in 19% of the cases, between 10 and 20 years of age in 37%, between 20 and 30

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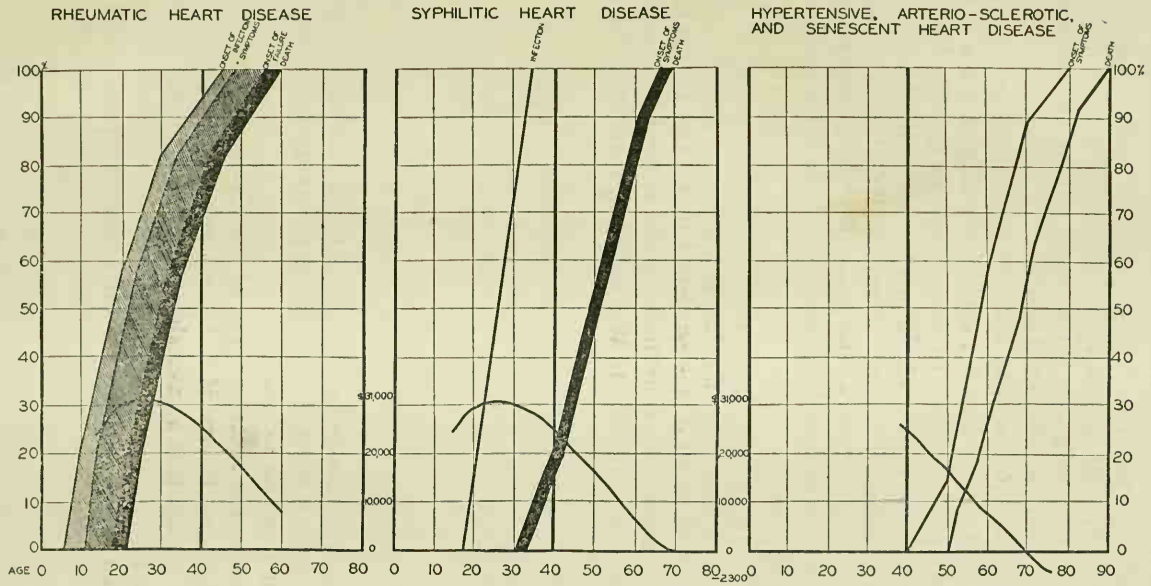
* "In this chart will be found graphically expressed the life history of the average heart patients of each of the three chief categories of heart disease across the span of life, as summarized from the literature by Cohn."†

† Cohn, Alfred E. Heart disease from the Point of View of the Public Health; Am. Heart Jour., Vol. II, Nos. 3 and 4, pp. 275-301, 386-407, Feb. and Apr., 1927."

COURSE OF HEART DISEASES
BY AGE OF ONSET, DISABILITY, AND DEATH, RELATED TO ESTIMATED LIFE VALUES



The onset of rheumatic heart disease occurs in sixty percent of the cases before the age of twenty years and in ninety-five percent before the age of forty. The period of time between infection and the development of disabling symptoms is usually longer in those attacked early in life than in those attacked after twenty, as shown by the varying width of the lightly shaded strip, averaging a little less than four years in duration.

Following this is a fairly uniform period of seven years (heavily shaded strip) during which there are disabling symptoms of varying severity but causing interference to marked degree, though intermittently with usual occupations.

Finally, the solid black strip expresses the period of about four years of increasing and almost continuously complete disability until death results from cardiac failure.

The curved line A represents the changing value of a life expressed in dollars, throughout the period of prevalence of rheumatic heart disease as indicated at the right hand of the graph.

The same use of lines and spaces prevails in the graphs of experience with syphilitic and hypertensive or senescent heart disease.

in 23%, and between 30 and 40 in 21% of the cases. Furthermore, we find on the average, a duration of four years between onset and partially disabling symptoms, which thereafter continue about 7 years before decompensation, and with it probably complete disability develops, which continues, alternating with partial and occasional working ability, for four years until death.

With syphilis, the infection which occurs between 18 and 30 years of age is followed, so far as heart disease including aortitis is concerned, by from 10 to 25 years without symptoms or conscious disability, death following usually within two years after the development of disabling symptoms. The deaths from syphilitic heart disease are therefore to be found in 80% of the cases after the age of forty, in 12% in persons over 60, and in about 34% of the cases each in the decades 40-49 and 50-59.

Of the 20% of syphilitic cardiac deaths occurring under 40 years of age, three out of four are in the decade between 30 and 40 years.

In the great mass of all deaths attributed to diseases of the heart, those of the senescent type, constituting 90% of the total, the onset of symptoms occurs (according to Wyckoff*) by decades as follows:

30-39	.4%
40-49	14.1
50-59	43.6
60-69	31.6
70 and over	10.2

This type of heart disease is not accompanied by any more or other disability than is commonly found among all persons in the later decades of life, the subjective symptoms of the disease rarely causing the patient serious occupational handicap unless he be alarmed by what he is told about his sickness.

In most cases in the senescent type of the disease, cardiac failure of a disabling degree occurs, if at all, so near to the time of death and at so late an age that a calculable period of economic disability does not develop.

Practically all deaths attributed to the senescent type of heart dis-

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* Wyckoff, John. Statistical Studies Bearing on Problems in Classification of Heart Diseases. II Etiology of Organic Disease. Am. Heart Jour., Vol. 1, No. 4, Apr., 1926.

ease occur after the age of fifty, their distribution among the subsequent decades of life being as follows:

Percent of all deaths from senescent heart disease falling in the decades

50-59 years	18%
60-69	29
70-79	33
80 and over	20
	100%

The following are typical composite histories from patients among the low income or dependent class, representing the three groups of heart disease leading to death, as assembled from the use of the detailed record forms, issued by the New York Heart Association.

Rheumatic Heart Disease.

White male patient, born in the United States of Italian parents. Reported at clinic at age 16. Patient had had rheumatic fever at the age of 11 and again at 14. At the age of 14 he began to have symptoms referable to his heart, dyspnea, and precordial pain. A tonsillectomy was performed at the age of 15. When patient was seen in the clinic at age of 16, the diagnosis of mitral insufficiency and stenosis was made. At the age of 18 he had his first attack of cardiac failure, with symptoms of dyspnea, orthopnea, precordial pain. He was cyanotic and his liver was palpable. The following year, at the age of 19, he had another attack of failure, more severe than the first. The patient died at the age of 21 of progressive congestive heart failure.

Syphilitic Heart Disease.

Male patient was admitted to the clinic at the age of 49. He had had gonorrhea at the age of 23 and syphilis at the age of 24. At the age of 48 he began to have cardiac symptoms, which progressed markedly until he died at the age of 50.

Arteriosclerotic Heart Disease.

Male patient was admitted to the clinic at the age of 55. At the age of 49 he began to have symptoms of heart failure but not of a degree of severity to interfere with work. The disabilities progressed until the time of his death, at the age of 57.

Estimates of the cost of heart disease in the United States can be approximated by calculating the losses due to premature deaths, and the costs of medical and nursing care, as in hospitals, dispensaries, by visiting nurses, in convalescent homes, and in homes and hospitals for chronic invalids.

Death Costs, 1928.

The population of the United States is estimated as of July 1st, 1928, to be a little over 120,000,000. The death rate from heart diseases (87-90) in the Registration Area in 1925 was 185.46 per 100,000 of the population. It is estimated that the deaths from heart diseases in 1928, if the rate of 1925 is not exceeded, will be 221,809. When distributed by age groups according to the distribution in per cent of heart deaths in 1925 and charging for the numbers occurring at each decade, the value of a life at the mid-period of the decade, we find the loss in terms of money value to be $1,538,897,680, or $12.65 per capita of the population.

Age Group	Estimated Distribution of Deaths from Heart Disease, 1928		Life Value at Mid-period Age		Loss to the Community
Under 10 years ..	1.6384	3,561	$14,156		$ 51,598,620
10-19	2.1414	4,673	25,341		120,724,524
20-29	2.5514	5,564	31,900		181,064,400
30-39	4.5166	10,015	28,750		288,851,250
40-49	8.5679	18,917	22,000		419,298,000
50-59	14.7041	32,493	12,900		421,946,100
60-69	23.4503	52,200	4,400		229,530,400
					$1,713,013,294
70-74	13.4982	30,027	— 766	$ 23,000,682	
75 and over	28.9317	64,359	—2,348	151,114,932	
					174,115,614
	100.0000	221,809			$1,538,897,680

While it may be quite justified from a purely financial point of view to calculate the loss to the community of the life of a woman on the basis of fifty per cent less monetary value than that of a man at the same age, there is a good reason to consider that the loss of life from heart diseases among women, decade by decade, and for equal numbers, constitutes quite as heavy a financial loss to the family and home in terms of dollars' worth of service and care as results from the same cause in the male. This is particularly the case in the economic group we are using as the basis of our estimates, i.e., persons earning $2,500 a year or less. Therefore, no attempt will be made to proportion the total cost of heart disease sickness and deaths according to sex incidence.

The death rate from heart disease in the registration area having increased 27.3% from 1920-25 (31% among males, 18% among females) it is certain that the 1928 estimate of deaths from the cause is conservative and well within the truth.

Hospital Costs.

In 1927 in the 4,322 general hospitals in the United States, 66% of the 345,364 beds were used on the average throughout the year, or 227,940. It was found* in 1921 in New York City, that while 4.6% of all general hospital patients are admitted for heart disease, 9.1% of the days of care provided in these hospitals are for this class of patients, or a total of 7,562,837 days of care for heart patients in the United States or, at an average cost of $4.00 a day per patient, a cost of $30,251,348. The average cost of hospital care in public general hospitals in New York State has in recent years (1926) been $3.08 per patient day, and in private or so-called endowed general hospitals $4.90 per patient day.

The experience of the State of New York including the City of New York shows a much smaller proportion of all hospital patients to be heart patients (1926†); i.e., 2.7% in the general hospitals of the State and City as compared with 4.6% in general hospitals in the city taken separately. If then the 2.7% of all general hospital patients in New York State and City are heart patients, and we have learned (1926) that the heart patient on the average uses about 1.74 times as many days of hospital care as other patients do, we shall find when applying this situation to the hospitals of the country as a whole that there would be 4.698% of total bed days of hospital use devoted to care of heart patients, or 3,904,319 days ($2.7 \times 1.74 \times 227{,}940 \times 365$), which at four dollars a day of care would be $15,617,276. The actual cost of hospital care for heart patients for the United States as a whole probably falls between these two figures; i.e., not over thirty nor under fifteen million dollars a year.

The reader familiar with the social practise of different parts of the United States as it affects the supply, use and per diem cost of hospital bed care for heart and other general medical patients will realize that estimates based on the experience in New York City and State may be in error, but information on the points at issue are not available from other large population groups. Also the fact that the reported death rate from heart diseases for New York State is higher than for any other state may result in an extent of hospi-

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* Emerson, Haven. The Chronic Disabled Heart Patient. Nation's Health, June, 1923.

† Halsey, Robt. H. Report of Committee to make a study of Heart Disease in the State of New York to State Med. Soc. May 1, 1928.

talization of this group of diseases above the average for the country as a whole. When more complete information is available the estimates offered here and now will have to be revised, but if present trends in death rates and hospital use and costs continue, the revision is likely to be upward rather than down.

It was found, furthermore, that 7.4% of the total cost of operating the public general hospitals in New York State, including New York City, in 1926 went for the care of heart patients, and 3.6% of the cost in the private general hospitals went for this purpose, the latter not being provided so commonly with beds available for the more chronic forms of disease. In the private hospitals in New York City the comparable figure was 4.3%.

Nursing Visit Costs.

Approximately 3,000,000 persons are in the boroughs of Manhattan, Bronx, and Richmond of New York City, within the reach of the Visiting Nurse Service (the Henry Street Nurses). Among these people 304,367 nursing visits were made to patients discharged from care in 1927, of which 7,229, or 2.4% were for heart patients. Stated in another way, these nurses visited 44,216 different patients in the year, of whom 692, or 1.6%, were heart patients, who received an average of 10.45 nursing visits each.

Among the entire population of the United States, approximately forty times the size of the community served by these visiting nurses, either visiting nurses or other persons with less skill and at more expense directly or indirectly, will be found to be providing some degree of home nursing, or a total of 289,160 individual services, which at the present actual average cost of visiting nurse service of $1.00 a visit would be be $289,160.

Cost of Clinical Care.

The six million people of New York City appear to be adequately served by the several heart clinics, as far as heart patients, suitable on economic and pathological basis for ambulatory clinic care, are concerned. Almost all ambulatory heart patients not under the care of private physicians go direct or by reference from other out-patient or dispensary classes to the special heart clinics scattered throughout the city.

The patients attending these heart clinics numbered 10,017 (4,592 adults, 5,425 children) and made 37,871 separate visits.* Of the total number of patients 22 adults and 211 children were found not to be suffering from heart disease.

Thus we find that there was one heart patient attending a heart clinic in the year for each 600 persons in the population and one clinic visit was made in the interest of heart disease by these patients for each 150 persons in the population.

It is known that as many as one-fourth of the entire population of the city calls upon the dispensaries for medical care in a year. We may reasonably estimate that there are four times as many ambulatory heart patients in the city receiving some medical care (three-fourths of them presumably at their own expense) as there are patients attending heart clinics.

Applying this ratio we should find that one person in 150 of the population of the country is a heart patient (800,000) capable of visiting a physician's private office or a public clinic and that there is made one visit by these heart patients each year for each 40 persons of the entire population.

At this ratio we should have about 3,000,000 visits a year, those at the public dispensaries costing the public and the patients about $1.00 a visit to provide, and the rest costing $2.00 or more for each visit, or approximately $750,000 for the dispensary visits and $4,450,000 for private physicians' care, or $5,200,000 for the population of the country as a whole.

The possible error of too high estimates based on experience in New York City is recognized, but final correction on the basis of morbidity records on a national basis must wait for the distant future. It must be remembered that the American heart movement had its origin in New York City and the more the knowledge of prevalence of heart disease is developed elsewhere the nearer do other communities approach the records of New York.

Convalescent Care Costs.

We have estimated (on the basis of New York City's experience) that there may be 7,562,837 days of hospital care given to heart

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* Summary of Clinic Reports, 1927, Com. on Cardiac Clinics. N. Y. Tuberculosis and Health Ass'n.

patients throughout the United States in a year, and about half as many if we base our calculations upon use of beds for heart patients in hospitals throughout the state outside of New York City.

In 1923 it was found* that in large general hospitals of New York City the average length of stay of heart patients was 52.2 days for each such patient admitted, the length of stay varying from 20 days at Bellevue Hospital to 140 days at the City Hospital for chronic invalids.

It was found in 1927† that the average length of stay of heart patients in 101 general hospitals in the State of New York was 24 days, varying from 14.3 days in Westchester County to 33.0 in Erie County, New York County giving 26.3. Taking the more conservative and probably more representative figure of 24 days care per patient, we can estimate on that basis, that there were somewhere between 160,000 and 315,000 heart patients admitted to general hospitals according to our use of state or city figures for the number of beds occupied by heart patients in general hospitals.

It has been found in various surveys of hospital and dispensary practice and from studies of the needs of patients as discharged from hospitals,‡ that among general medical and surgical patients and maternity patients about 10% needed as much as three weeks convalescent care. More nearly twice as many of hospital heart patients require such a convalescent period as do the general run of patients, and for a six weeks period instead of three weeks.

Similarly, it is found that, while of general dispensary patients some two per cent need convalescent care for three weeks, probably at least 4% of heart patients attending clinics need such care for as much as six weeks.

Twenty per cent of hospital heart patients would be somewhere between 32,000 and 63,000 according to our basis of estimating heart patients as above.

Four per cent of the probable one-fourth of the ambulatory heart patients attending clinics (200,000 as given above) would be 8,000.

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* Emerson, Haven, and Woughter, M. L. Chronic Disabled Heart Patient, Nation's Health, June, 1923, Vol. V, No. 6.

† Halsey, Robert H. Report of Committee to make a Study of Heart Disease in the State of New York to State Med. Soc. May 21, 1928.

‡ Cleveland Hospital and Health Survey, 1920, Convalescent Care, Frederic Brush, M.D., p. 939.

Six weeks convalescent care for these two groups of patients would amount to somewhere between 1,680,000 and 2,982,000 days of convalescent care.

This type of care costs about half as much per diem as does general hospital care, so the expense of such care if provided would be between $5,964,000 and $3,360,000.

We do not know the number of days of care provided for heart patients in convalescent homes, but we do know (Bryant, Convalescence, 1927, pp. 255-257) that there are about 12,000 beds devoted to convalescent care for part or all of the year outside of the vicinity of New York City and about 3,000 more in the New York City environs. Many of these beds are available for only a few months each summer. Others, though intended for use throughout the year, are not always occupied, and there is no reliable record of the number of bed days of convalescent care provided for heart patients.

However, since New York City's 6,000,000 people are pretty well served in respect to convalescent care for heart patients by the 370 beds in use for this purpose throughout the year and used to a high percentage of their capacity, we might expect twenty times as many beds to be needed for the country as a whole or 7,400, which falls between the figures given above, 1,680,000 days of care calling for 4,602 beds and 2,980,000 days requiring 8,164 beds. Our estimates of cost are then within reasonable limits.

It is fully recognized that no such amount of care for convalescent heart patients is at present provided for in institutions operated for this purpose. However, if heart patients do not have such care in institutions or its equivalent under individual conditions, they will suffer avoidable damage from lack of it. Therefore, this item is added to the total estimate as a proper charge.

Chronic Sick Costs.

In a survey of chronic sickness, recently completed in Boston,* it was found that one person among every 185 of the population was a chronic invalid and that 19.6% of these were heart patients, of whom 30.7% were persons under twenty years of age and 42.01% were persons under 40 years of age. All but 30% of all chronic heart

* Chronic Disease Survey of Boston, 1928, by Amy Hamburger, Council of Social Agencies, Boston.

patients were found to need care throughout the year, and it was further found that this was generally provided at about one-half the cost per patient day of the cost of care in general hospitals.

On this basis applied to the United States as a whole, the estimated 89,122 chronic heart patients needing care the year round are probably costing in the neighborhood of $65,059,060 a year. (120,000,000 ÷ 185 × 19.5% × 70% = 89.122. 89.122 × 365 × $2.00 = $65,059,060.00.)

Assembling our variously estimated items of the cost of the medical and nursing care of heart patients in the United States in this year, we have in round numbers for those:

A	Under care in general hospitals	$15,617,000	to	$30,251,000
B	Under care by nursing visits in homes	289,000		
C	Attending clinics or physicians' offices	5,200,000		
D	Under care in convalescence	3,360,000	to	5,964,000
E	Under care in chronic stage	65,059,000		
	Total cost for a year	$89,525,000	to	$106,763,000

Or we might properly say that we have estimated the above costs on the basis of a total of 1,148,800 to 1,179,800 heart patients of the various categories in a current given year or just under one per cent of the population as follows:

A	Under care in general hospitals	$160,000	to	$315,000
B	Under care by nursing visits in homes	27,680		
C	Attending clinics or physicians' offices	800,000		
D	Under care in convalescence	232,000	to	263,000
E	Under care in chronic stage	89,120		
		$1,148,800	to	$1,179,800

Estimates have ranged from one to two per cent of the population as constituting the total of heart patients.

Using one per cent of the population as being nearer our own estimate, built up from the various groups probably receiving care, and using the lower of our total cost figures ($89,525,000), we find that the average heart patient in the United States to-day probably carries an annual charge of $74.60 or we might properly say that heart disease puts a burden of about 75 cents on each person of the entire population each year.

The cost of heart disease deaths occurring in the various decades of life was found to be $1,538,897,680 each year, or a loss of $12.82 for each member of the population.

In 1922 at the then death rate from tuberculosis in the United States, it was estimated* that the shortening of life by this disease, which amounted at that time to a reduction of the average length of life of all the people of the country by 2.5 years each, would cost the entire population of the country at that time during their complete life span the sum of $27,125,000,000.

On the basis that in 1928 heart diseases will cause a reduction in the average length of life of people in the United States of 1.66 years for males and 2.00 years for females, the cost to those now living in the United States from the shortening of lives by heart disease will amount to $21,960,000,000 during their life span.

While heart diseases cause more deaths than tuberculosis, the age of death for the latter is much earlier, hence the greater effect of tuberculosis deaths on the average length of life, and on the cost of tuberculosis to the population as a whole.

It has been estimated recently by Louis I. Dublin† that the loss each year, on the basis of 1927 experience, due to the shortening of life by cancer, amounts to about $680,000,000 which may be compared with the estimates for the population of 1928 on the basis of the 1925 heart disease death rate, as presented above, $1,502,198,355, the greater annual cost from heart disease deaths being due to their greater frequency in the population, even though the largest number of deaths from this cause in any one decade of life is in a later decade than is the case with cancer deaths.

It was estimated on the experience of 1922 in the United States that the annual cost of tuberculosis patients was probably about $3.15 per capita of the population. It has just been shown that for heart patients the entire population must bear a cost of 75 cents per capita per annum. This difference in the cost is probably due to at least two important factors: namely, the greater adequacy of provision for the medical and nursing supervision of tuberculosis as compared with those available for heart patients, and the added costs of sanitary supervision and isolation of many tuberculosis patients, of a kind not required for heart patients, except for those in certain stages of acute rheumatic fever and its recrudescences.

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* Haven Emerson. A Brief for Investment in Adequate Prevention of Tuberculosis, Amer. Review of Tuberculosis, Vol. VI, No. 6, Aug., 1922.

† Dublin, L. I. The Money Loss from Cancer, Campaign Notes, Amer. Soc. Control Cancer, 1928, July.

Probably some postponement in the age of death from heart disease of rheumatic origin and possibly some reduction in the incidence of acute rheumatic fever might result from an expenditure per patient, or per capita of population, for heart disease more nearly equal to the sums found desirable for the prevention of tuberculosis and the treatment of the tuberculosis patient.

The burden of heart disease falls more heavily, absolutely in terms of incidence and deaths, and relatively in terms of loss of livelihood, upon the unskilled wage-earner, the under-privileged man and woman, than upon any of the higher earning or social levels of the community.

Because of its duration, heart disease, with the exception of tuberculosis, mental alienation and certain forms of chronic arthritis, constitutes heavier burdens upon wage earners than do other diseases, this burden increasing with each decade of life from twenty-five to sixty-five.

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THE HEART IN INFECTION.

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The subject assigned for this paper recognizes that heart disease is rarely an affection of the heart exclusively, but rather only a part of a general systemic malady. In both its anatomy and physiology the heart has unique features which require consideration, but too close attention to them without due regard to the general problems of the biology of infection and degeneration may have a deterring rather than a beneficial effect.

It has become customary to divide heart diseases roughly into two general types: infectious and degenerative. The first are thought to exert their baneful influence largely during the first four decades of life, and the second class during the remainder. Vital statistics, indeed, confirm this impression; and a study of the form and function of tissues during the afternoon of life leaves little doubt of the importance of the alterations normally occurring in senescence as playing an important rôle in the production of cardiac diseases. But the age-linked features of these later alterations in structure may have blinded us somewhat to the relationship between infection and degeneration. Indeed some recognition of this relationship has been gained from the construction of actuary statistics, where their proper evaluation is a matter of dollars and cents. But only as the study of pathology becomes more and more a correlation of distant as well as immediate cause and effect, will there emerge a recognition of the true relationship of infection during the early years of life to late degenerations.

The effect of any infection upon the heart depends upon at least two factors: the nature of the infection and the reaction of the heart to that particular infection. The heart, being a complex organ, in turn shows different effects according as one or another of its component parts is involved. The heart, moreover, suffers a double insult both as a direct result of bacterial injury and as an indirect result of disturbed circulation; hence a vicious circle is quickly established in a cardiac infection. We must, therefore, keep these complicating factors in mind, for the result of even the simplest

injury from bacteria is much more complicated than we imagine when we reduce the various elements to simple terms.

Again, we must recognize our comparative ignorance as to how the myocardium reacts to noxious agents. For example, we do not know to what extent or in what direction edema *per se* influences the various functions of the heart muscle: Does it increase or decrease irritability, contractility, or conductivity? Does it increase one and decrease another? What is the synergic or antagonistic result of these factors? Does mild prolonged irritation from infection lead to hypertrophy, or is hypertrophy only a response to a demand for increased work? Until we are able to give a positive answer to many such questions our discussions will, necessarily, have much of a hypothetical character.

We are in possession of enough data, however, to sketch at least rough outlines of many features of infectious heart diseases. At times it is necessary to obtain information concerning some factors from a direct study of infectious agents in parts of the body permitting direct examination, hence more accurate analysis. We must also resort to information gained from experimentation on lower animals, though in this respect, it must be confessed, we have obtained relatively little direct knowledge, in so far as infection of the heart is concerned. Aside from the type of heart involvement found in diphtheria and that of subacute bacterial endocarditis, we are unable to reproduce with any degree of regularity in lower animals the various pictures of cardiac infection that we encounter in man. Comparative experimental investigations, therefore, have yielded more direct information from the standpoint of physiology than from that of infection.

Keeping reservations of this nature in mind, we shall base our discussion upon types of reaction in certain infections, rather than attempt to cover all of the features of infectious heart disease. A satisfactory picture of the various forms of cardiac injury from bacteria can probably be most clearly presented by using as examples the following diseases: diphtheria, scarlet fever, rheumatic fever, subacute bacterial endocarditis, syphilis and septicopyemia.

Diphtheria.

In diphtheria the simplest picture of cardiac injury is seen. The heart is poisoned by the toxin produced at a distance, for it has been

shown that all of the characteristic histological alterations found in the heart in fatal cases of diphtheria can be reproduced experimentally by injecting animals with bacteria-free diphtheria toxin.

The essential alteration is a degeneration of the muscle fibers, variously described as waxy, hyaline, and fatty; the differences recorded could no doubt be reconciled if variations in strength of toxin and time of action were considered. But injury to the parenchyma is quickly followed by interstitial response with edema, accumulations of wandering cells and proliferation of fixed connective tissue elements. All of this secondary picture may be considered an effort to remove the debris of the irreparably damaged parenchyma and to repair the injury in so far as possible. Warthin (1) describes muscle regeneration beginning in the degenerated muscle fibers, and attributes the apparently efficient functional recovery seen in most cases of diphtheritic carditis to a fairly complete regeneration of the myocardium.

While the endocardium is grossly normal and verrucous endocarditis is rarely, if ever, a complication of diphtheria, both Baldassari (2) and Czirer (3) have described focal lesions in the endocardial and subendocardial tissues of the mitral valves. Even though endocardial injuries occur, it is evident that recovery usually takes place without permanent damage to these structures. Indeed, one of the most striking peculiarities of the diphtheritic heart is the disproportion between the degree of functional disturbance at the time of the acute attack and the small amount of residual organic or functional crippling (4). During the acute stages the injury is made evident by marked myocardial weakness, and also by various types of cardiac irregularity, which in recent times have been proved to be due to ectopic beats, nodal rhythm, heart block, and occasionally to auricular fibrillation. Probably in no other disease excepting rheumatic fever is it possible to demonstrate involvement of the conduction system so frequently by physiological methods of investigation. Histopathological proof of such involvement has also been adduced.

The patchy distribution of the degeneration which has been demonstrated histopathologically explains the possibility of recovery, for enough efficiently functioning myocardium usually remains to permit life to continue until reparative processes have progressed to a point where normal bodily function may be resumed. Even in severely poisoned hearts Edmonds and Cooper (5) have demonstrated that the muscle and conduction system can respond in a normal man-

ner to drugs. The ultimate outcome in any given case doubtless depends upon the ratio of uninjured to injured muscle; and the difficulty encountered at the bedside is to estimate this ratio, for upon it rests both treatment and prognosis.

Scarlet Fever.

The different cardiac complications of scarlet fever, although comparatively infrequent, illustrate the various forms of cardiac injury resulting from infection more clearly than does any other single disease. For purposes of discussion these complications may be classified as (1) toxic; (2) allergic; (3) septicopyemic. The toxic cardiac symptoms closely resemble those found in diphtheria. They come on shortly after the initial defervescence, are often accompanied by bradycardia, and are characterized by signs of cardiac weakness. Usually there is little if any fever and very mild evidence of general intoxication. If the patient remains flat in bed he may give slight, if any, evidence of cardiac weakness, while, on the other hand, if he assumes the sitting or standing posture there occur dizziness, weakness and other signs of an insufficient heart. Escherich and Schick (6), who designate this condition as myasthenic, state that it practically never has a fatal outcome; hence histopathological descriptions are lacking. The similarity of the early symptoms of scarlet fever to those of diphtheria and the possibility of neutralization of the so-called scarlatinal toxin with an antitoxic serum, moreover, make plausible the hypothesis that this peculiar myasthenia cordis is due to a direct toxic action of a poison on the heart muscle.

The cardiac complication usually placed among the allergic manifestations of the disease is verrucous endocarditis, probably better called valvulitis. Schick (6) advanced the theory that the complications occurring from the end of the second to the sixth week after the onset of scarlet fever and with greatest frequency about the twentieth day are allergic in nature. This group includes adenopathy, recurring angina, nephritis, arthritis and occasionally simple endocarditis; they are always accompanied by fever. Nicoll (7) states that endocarditis is practically always a complication of arthritis and designates it a "complication of a complication." The assumption that both are manifestations of a common causative condition would, however, be more logical. Escherich and Schick attribute all of these symptoms to an allergic state which develops as the result of the

prolonged action of the causative agent of scarlet fever in the body. At the time of their publication the etiological rôle of the streptococcus scarlatinae was not established, but to-day we can assume with a fair degree of assurance that the stimulus to the development of this hypersensitive state is a streptococcus.

Hitchcock and I have been able to induce a condition of marked hypersensitiveness in rabbits by inoculating them intracutaneously with minute doses of hemolytic streptococci. Zinsser and Grinnell (8), and Mackie and McLachlen (9) have sensitized guinea pigs with both toxin and intact cells of streptococcus scarlatinae. Dochez and Stevens (10) have furthermore shown that two types of hypersensitiveness can be induced in rabbits by repeated injections over long periods with streptococcus erysipelatis toxin. The first to appear is neutralizable with an antitoxic serum; the second is not so neutralizable, and when present can be demonstrated by testing with filtrates from a variety of streptococci. Their work can be brought into relationship with both early and late clinical manifestations of scarlet fever. The early symptoms may be considered as evidence of hypersensitiveness to the toxin; the later ones as hypersensitiveness to the whole bacteria or to some other bacterial product. A series of scarlet fever patients should, however, be tested from this point of view before it can be definitely established.

Another hypothetical angle from which to view scarlatinal carditis is that these late allergic phenomena occur in those scarlet fever patients who are already partially hypersensitive to streptococci and that the added stimulus resulting from the scarlatinal infection increases the already existing allergy to a point where clinical symptoms are elicited. The well known liability of a patient with quiescent rheumatic endocarditis to develop a relapse of rheumatic carditis following an attack of scarlet fever could be explained on these grounds. This liability has been recently emphasized by Hector (11).

Since Romberg's (12) histopathological description of myocarditis in the different infectious diseases there has been much discussion of the relation of these structural changes to the clinical symptoms, and the heart in scarlet fever has not escaped consideration. The resemblance between late sequelae of scarlet fever and of rheumatic fever has directed the attention of pathologists towards a comparison of the microscopic picture in the two diseases. Schmorl (13) in 1914 stated that he had found lesions closely resembling Aschoff bodies in the heart of a scarlet fever patient. Stimulated by this finding,

Fahr (14) made a detailed study of the myocardium in eight fatal cases of scarlet fever, as a result of which he concluded that the myocardial alterations in this disease resemble those of rheumatic fever more closely than they did those of any other infectious disease. He described them as perivascular granulomas, made up of proliferative cells from the perivascular adventitia; cells which were basophilic and stained red with pyronin. No giant cells were found, but myolysis of neighboring muscle fibers was often present. Thus, absence of giant cells and smaller size of the lesions were the chief features differentiating these lesions from those of rheumatic myocarditis. In certain cases of rheumatic carditis, on the other hand, interstitial myocardial foci were found indistinguishable from those of scarlet fever. Fahr mentions that the age of the lesions may be of significance but leaves the determination of this question open for future studies. He states that the valves in these hearts were normal but that focal thickening of the mural endocardium was present in four out of nine cases. Czirer (3), on the other hand, described microscopic focal valvular lesions in thirteen cases of scarlet fever, and attributed later changes often found at autopsy to these earlier focal injuries. While it is evident that the whole subject requires additional study, these investigations indicate the many elements of similarity between the heart in scarlet and rheumatic fevers.

The septicopyemic type of cardiac complication occurs in those rare instances where the streptococcus scarlatinae becomes implanted and grows on the heart valve. It is an accident comparable to other forms of malignant endocarditis, and only related to scarlet fever because hemolytic streptococci in general have the capacity for producing this type of valvular disease.

Rheumatism.

Heart involvement in rheumatic fever has interested pathologists and clinicians for over a century. Early the close relationship between verrucous endocarditis, pericarditis and what was then called acute articular rheumatism was well established, and the relationship of acute rheumatic endocarditis to chronic valvular disease was fixed. Following the description by Aschoff (15) and by Geipel (16) of the peculiar submiliary nodules in the myocardium which now bear the former's name, the attention of both pathologists and clinicians was turned chiefly towards the myocardium and con-

duction system. But in recent years with a more extensive study of the general pathology of the disease, we are realizing that the pictures simply of the myocardial Aschoff body and of verrucous endocarditis are not sufficient to explain all of the damage caused by rheumatic fever. Indeed, we know that they represent only two reactions, to which other types of tissue response must be added if we are to have a satisfactory conception of the pathological evolution and functional disturbance seen at various periods of the disease.

Even before Aschoff's discovery of the specific submiliary nodule the Leipzig school (17) had emphasized the importance of interstitial myocarditis in causing the symptoms referable to the heart in various common infectious diseases; but Aschoff and Tawara (18) a few years later strongly refuted the argument that all of the symptoms of myocardial weakness could be explained on the basis of the interstitial myocarditis in typhoid fever and scarlet fever, or by the presence of submiliary nodules in the rheumatic heart. In this same period the views of Poynton and Paine (19) concerning the etiological rôle of the streptococcus rheumaticus were being advanced; and simultaneously numerous observers, among whom Schottmüller (20) and Libman (21) were most prominent, were establishing definitely the rôle of various strains of non-hemolytic streptococci in the production of subacute bacterial endocarditis. In the meantime physiological methods had claimed the attention of most investigators in the study of heart disease, and because in some instances a correlation of certain definite functional disturbances with lesions of the conduction system could be shown there arose a hope that most of the problems of cardiac disease were to be solved by the application of physiological and pathological data already available. But with the passing of years came the realization that much more information was required before a solution of the problem was in sight; and recently a large number of investigators have been directing their attention to various phases of rheumatic heart disease.

So far as we can determine, the earlier proponents of the etiological rôle of non-hemolytic streptococci in rheumatic fever never advanced a satisfactory hypothesis to explain the difference in the clinical and pathological pictures seen in this disease and that of subacute bacterial endocarditis. In fact most of the experimental evidence pointed to the ease of producing experimentally subacute bacterial endocarditis and the difficulty of reproducing any tissue changes at all comparable to those found in rheumatic fever patients. The

diverse cultural and immunological characteristics of the strains of streptococci recovered by different workers added to the difficulty of bringing them into causative relationship with a disease which seemed to be such a definite clinical entity.

In our own clinical studies we had become more and more impressed with many points of resemblance between rheumatic fever, tuberculosis and syphilis, and hence suggested an allergic explanation (22). Shortly afterwards Bezançon and Weil (23) independently advanced similar views. We applied methods which had proved useful in the study of the last two diseases to try to find some new etiological agent comparable to the tubercle bacillus or the *Treponema pallidum*. Many species of laboratory animals, including calves, were inoculated in various manners without successful reproduction of anything closely resembling rheumatic fever and without demonstration of any new etiological agent. The only microorganisms recovered with any degree of regularity from blood cultures or subcutaneous nodules were streptococci.

In the past four years, however, in collaboration with Drs. Andrewes, Derick and Hitchcock, we have obtained experimental data with these cocci which appear to have brought some order out of what was previously a confusing situation. The detailed evidence for our work is published elsewhere (24), (25), (26), (27), (28), (29), (30). It is sufficient to state here that by intracutaneous inoculation with certains strains of streptococci we have been able to induce in rabbits a general state of hypersensitiveness which resembles in many particulars the allergic state seen in tuberculosis and syphilis. A most important requirement for the induction of this state is the production of a focal lesion somewhere in the body. Lesions repeatedly produced at suitable intervals increase the hypersensitiveness. Once induced it can often be maintained over comparatively long periods by making a deep focus of suitably infected agar.

The tissue of a highly hypersensitive animal shows a greatly intensified response to inoculation with very small amounts of streptococci; for example, where a normal animal shows little or no reaction to 0.0001 cc. of broth culture, the hypersensitive animal has a lesion containing 100 to 400 cubic millimeters of exudate and proliferate; and a highly sensitized animal at times shows an edematous lesion 10 to 12 mm. in diameter following inoculation of 0.000001 cc., while a normal animal shows only a needle prick. Microscopically the lesion of a hypersensitive animal shows early a much more marked

exudation than is seen in the lesion produced by the same size inoculum in a normal animal. But a point of almost as great interest is that an intravenously inoculated rabbit does not show hypersensitiveness of this type. On the contrary, with repeated intravenous immunization it shows less and less reaction to intracutaneous test inoculation; and these cutaneous lesions in an immune animal show less exudation microscopically than does a normal control. We thus have two types of tissue response to non-hemolytic streptococci developed according to the manner in which the animal is originally inoculated: hypersensitive, following inoculation into the tissues, and immune, following intravenous inoculation.

But these two states are not specific when specificity is considered from the viewpoint of cultural or serological strain specificity. While a hypersensitive animal usually reacts more strongly to the strain with which it is sensitized, it also over-reacts to more distantly related strains. Likewise, an intravenously inoculated animal shows the immune type of tissue response to subsequent intracutaneous inoculation with heterologous strains. A similar observation was made by Kuczynski and Wolff (31), who observed that intravenous immunization of mice with green streptococci markedly increased their resistance to hemolytic streptococci, and by Tillett (32), who showed that active immunization with rough pneumococci rendered a rabbit immune to all types of pneumococci.

We have, furthermore, been able to carry some of these studies over into the clinic, and to show that most patients with rheumatic fever show much larger reactions following intracutaneous inoculations of vaccines or of nucleoproteins prepared from streptococci, and of filtrate of non-hemolytic streptococci, than do normal people. The observations of Birkhaug (33), Kaiser (34), Small (35, 36, 37), and Strauss (38), also indicate similar hypersensitiveness in the rheumatic fever patients they studied.

In one respect our observations differ from Birkhaug's, viz., that patients with active rheumatic fever show skin hypersensitiveness to filtrates of various types of non-hemolytic streptococci.[1] Mackenzie and Hanger's (39) observations indicate also that the specificity of skin reaction is not linked to any one type of streptococcus, nor in their observations was any one type of disease linked with hypersensitiveness to streptococci. While they did not give their complete

[1] Birkhaug. Personal communication has confirmed our observation.

data, it is interesting to note that many of their hypersensitive patients were suffering from rheumatic fever.

We (28) have furthermore observed that rheumatic fever patients usually show a late tuberculin-like febrile reaction following intravenous injection with small doses of vaccine or of nucleoprotein prepared from hemolytic streptococci. While we do not claim that rheumatic fever is the only condition in which these reactions are found, still we feel that this kind of hypersensitiveness plays an important rôle in producing the peculiar picture of the disease. Zinsser (40) on hypothetical grounds has recently advanced similar views. Indeed, we are appreciating more and more that it is the peculiarity of the tissue response rather than a history of polyarthritis that is the important factor. The recently published statistics of von Glahn (41) emphasize the frequency with which rheumatic carditis may occur in the absence of a history of polyarthritis.

In the production of most diseases a number of contributory factors must be present. As yet we are unable to state why all persons hypersensitive to streptococci do not have rheumatic carditis, any more than we can determine why all scarlet fever patients do not develop nephritis, or even why some with streptococcus scarlatinae infections do not have a rash; only with the acquisition of much additional information can we furnish completely satisfactory answers to these questions. But the existence of a state of hypersensitiveness to streptococci can account for many of the known features of the disease. The lack of strain specificity of this state could easily make understandable the etiological rôle of the several types of streptococci that have been recovered from patients with this disease. The extensive exudative reaction seen in hypersensitive rabbits following implantation of small numbers of streptococci explains how such relatively avirulent micro-organisms could produce extensive edema and marked outpouring of cells in patients who are similarly hypersensitive.

For a period following Aschoff's discovery of the submiliary nodule, which seemed to be chiefly a proliferative reaction, pathologists neglected the exudative reaction which is so evident to clinicians in the swelling seen in polyarthritis; but lately the studies of von Glahn (42) and Pappenheimer (43), MacCallum (44), and Fahr (14), and our own observations (45) have directed attention to the importance of exudation as a part of the tissue reaction. If we can assume that the submiliary nodule represents mainly the proliferative

response of the tissues to implantation of bacterial body or some particulate product of such bacterial body, then it can be readily understood how such an exudative response as edema might occur at some distance and lead to distinct clinical symptoms. The disappearance of the exudation about joints following the administration of salicylates is a common experience; a similar decrease of exudation in the heart is the most plausible explanation of the recent observations of Levy and Turner (46) who demonstrated the disappearance after administration of salicylates of abnormally prolonged conduction time in several patients with rheumatic fever. The occurrence of such an exudation, which may appear and disappear with surprising rapidity, would also explain in part the frequency with which electrocardiographic evidence of cardiac involvement occurs and the numerous changes that may be demonstrated in a single patient.

In our opinion, edema of the valves may account for the localization of verrucae at the line of closure. Elsewhere (22), (47) we have called attention to the occurrence of widespread interstitial valvulitis early in the disease, in some instances before any verrucae were demonstrable. If edema in valves approximates in intensity that of joints, it is easy to understand how such an edematous structure impinging on another similar structure thousands of times a day would lead to injury to the endocardium sufficient to favor the deposition of thrombotic deposits in the form of verrucae. Coombs (48) also considers that interstitial valvulitis is the essential rheumatic valvular lesion.

But in this connection we must consider the recent work of Siegmund (49), Dietrich (50), and Kuczynski and Wolff (31). Siegmund, particularly, has studied the reaction of vascular endothelium to infection and shown that this endothelium reacts quickly following intravenous inoculation with various bacteria. Small intimal "Fibrinknotchen" are formed in which the bacteria are quickly destroyed; but during the evolution and involution of these small intimal nodules various pictures are reproduced which closely resemble certain features of endocarditis. With increasing immunity there is increasing intensity of response. His findings can be readily brought into relationship with subacute bacterial endocarditis, but he did not consider the possible differences between a hypersensitive and an immune state in his animals, such as we have been able to reproduce.

Dietrich, on the other hand, suggests that increased sensitivity may

account for the implantation of bacteria on the margins of the valves. He inoculated two series of rabbits intraperitoneally with staphylococci, and one series intracutaneously by rubbing broth cultures into the shaven skin. Subsequent intravenous inoculation with cultures of low virulence resulted in the formation of verrucous lesions along the valve margin in a number of instances. Unfortunately, a microscopic examination of these lesions was not given.

In all of the work of the German school the extension of the infection of the valve from the external surface inward is stressed. In this country, on the other hand, the weight of the evidence is thought to indicate that infection of the valve occurs through the vessels supplying the valve. The work of Kugel and Gross (51), of the students of Winternitz (52), of Kerr (53), of Bayne-Jones (54), and others, all show that vascularization of the valves is much more extensive than was formerly known. Both Gross and Winternitz have found that endocarditis and vascularization of the mitral valve run hand in hand, and feel that all of the evidence points to such vascularization as being primary. It is interesting to note that endocarditis occurs most frequently in rheumatic fever of children at an age when vessels are most numerous, and that liability to endocardial involvement decreases with age in about the same ratio as vessels are known to disappear; also that frequency of involvement of the respective valves is in direct ratio to demonstrated frequency of vascularization. It is a general rule that localization of rheumatic fever lesions occurs most readily in dense connective tissue where there is constant motion. The valves represent such a tissue. If hypersensitiveness to streptococci of the vascular and perivascular tissue of the valves of a rheumatic fever subject is at all comparable to that of his subcutaneous tissues, or to that of the hypersensitive rabbit, it is readily understandable how interstitial valvulitis might arise from implantation of streptococci or their products in these vessels when the individual is hypersensitive. Indeed, we have observed distinct endo- and perivascular reactions in small blood vessels in the valves comparable to similar lesions in the synovial and perivascular tissue removed by biopsy from the knee joint of patients with active rheumatic fever.

The reaction in the vessel wall may indeed play a larger part in the production of other cardiac disturbances than we have heretofore considered probable. Fahr (14) called attention to the frequency of some form of vascular lesion in the majority of 32 cases

of rheumatic carditis which he examined, and suggested that these lesions would account for areas of transient or partial anemia in the heart wall which in turn would explain the muscular weakness which is often so prominent a clinical feature. He goes even farther and suggests that similar vascular alterations may account for some cases of malignant sclerosis. Von Glahn and Pappenheimer (55), (56) have still more recently directed attention to the widespread distribution of vascular lesions in rheumatic fever; and Kugel and Epstein (57) have just contributed similar valuable additional evidence. Winternitz and his students (52) are actively studying the effect of lesions of the vasa vasorum and pointing out the importance of these lesions in disease of the larger arteries and veins in infection. When all of the work at present in progress is correlated we may find that the so-called rheumatic fever reaction has a wider import than we even now think probable. Indeed, the conception of rheumatic fever as a joint disease is becoming less and less prominent as its rôle in contributing to disease of visceral blood vessels becomes more evident.

Subacute Bacterial Endocarditis.

It is desirable next to discuss the disease known as streptococcal subacute bacterial endocarditis or endocarditis lenta from the standpoint of recent investigations. Both Small (35) and Birkhaug (33) have recently directed attention to the frequency with which rabbits develop a disease somewhat similar to subacute bacterial endocarditis when inoculated intravenously with indifferent streptococci, and we have confirmed these observations. Rosenow (58) obtained similar lesions by injecting green streptococci. But in human pathology this bacterial endocarditis is practically always secondary; in other words, the bacteria are implanted on or in an abnormal valve, and in the majority of instances the valve has been the site of an old rheumatic endocarditis.

Another interesting observation originally made by Kinsella (59) and myself, and confirmed by Libman (60), is that all of these patients show a fairly high agglutinin titer in their serum against the streptococcus which is producing their disease. Kinsella (61) was unable to produce endocarditis regularly on the traumatized valves of dogs' hearts unless he previously partially immunized the animals. Wright (62) was able to produce bacterial endocarditis more regularly in rabbits if they were previously immunized. Wadsworth

(63) has directed attention to the frequency with which horses highly immune to pneumococci develop endocarditis resembling subacute bacterial endocarditis in man. White (64) has recently observed a similar condition in streptococcus immune horses. Serum immunity and this type of endocarditis seem then to go hand in hand. We have never found a similar high degree of serum immunity in rheumatic fever patients against the strain of streptococci occasionally recovered from them. This, moreover, corresponds with our findings in hypersensitive rabbits, where agglutinins and precipitins against the sensitizing streptococcus are never very strong. Small (35), (36) and Clawson (65) both report the finding of weak agglutinins against the streptococci which they recovered from patients with rheumatic fever, but here again, let us emphasize, the titer was low. In this connection another observation made by Howells and Corrigan (66) is of much interest. These authors found that none of seven patients with subacute bacterial endocarditis showed skin reactions to filtrates of streptococci, and we have confirmed these findings in two instances. Kinsella and Garcia (67) also were unable to induce any local reaction by inoculating a patient suffering from subacute bacterial endocarditis with living streptococci, or by making an agar focus inoculated with these streptococci. This is directly in line with our observations that intravenously immunized rabbits show less intense reactions than do normal animals to cutaneous inoculation with streptococci or with filtrates. What more do we find clinically? Practically none of the embolic phenomena of subacute bacterial endocarditis are accompanied by extensive exudation; but the evidence of tissue injury is closely confined to the area of the embolus. We have never seen marked edema about a painful joint in the neighborhood of Osler's nodules, nor about a vessel in the lobe of the ear where we could observe the relationship of the embolus to the blood vessel. We have recently observed a patient with this type of endocarditis involving chiefly the right heart, but in spite of numerous pulmonary emboli no pleural effusion occurred. Another patient recently under our care entered the hospital with rheumatic fever and during the next two months had all of the exudative phenomena usually seen with pericarditis, pleurisy and polyarthritis. He then seemed to recover but after an acute sinusitis developed classical signs of subacute bacterial endocarditis, and showed *Streptococcus viridans* repeatedly in blood cultures. His blood which had been saved from the period when he was suffering from polyarthritis showed no agglutinins, while that obtained after

the development of subacute bacterial endocarditis showed them in a fairly high titer. No exudative phenomena were observed during this latter period.

The interesting observations of Rothschild, Sachs and Libman (68) may also be added to the evidence concerning the relationship of this disease and rheumatic fever. In 61 cases of bacterial endocarditis examined by graphic methods they found abnormal increase of the conduction time only ten times and changes in the Q R S T complex of the electrocardiogram only four times. This, compared with lengthened P R interval in 23 out of 65 cases of rheumatic fever and alterations in the Q R S T complex in 52 cases, and with similar findings by us (69), shows that instrumental signs of extensive parenchymatous irritation of the heart are as rare in subacute bacterial endocarditis as are peripheral signs of exudation.

From all of this evidence we may conclude that, in general, subacute bacterial endocarditis is accompanied by a state of relative tissue immunity, and that the presence of antibodies in the serum under experimental conditions is a concomitant phenomenon in the course of this disease. We then have the apparent paradox of an active and usually fatal infection in a biologically immune individual. How the micro-organisms originally colonize in or on the valve is a mystery. One possible explanation suggests itself: the streptococci in the blood stream of an immune individual may be agglutinated in masses of sufficient size so that when caught in a tissue, such as that of the heart valve, in which the natural factors for resistance are but slowly mobilized, they are large enough to start the growth of a colony; and this colony deep in the tissues soon increases sufficiently to remove many of its members from the influence of either tissue or circulating phagocytes. The blood of these patients is highly bactericidal to the infecting organisms; hence the only possible explanation for the continuation of the disease is that the bacteria are in a situation where this bactericidal action cannot be exerted. If this explanation is correct, the failure to effect a cure by attempting actively or passively to increase the immunity of patients suffering from this disease is understandable. All of the conditions for effective general immunization are present in such a patient. The etiological agent is being constantly supplied to the tissues with a consequent immune response. In rare instances this response is doubtless effective, but in most patients the streptococci are situated so that no immunity either active or passive can be made sufficiently

intense to destroy them. Chemo-therapeutic measures have been equally ineffective, and probably for similar reasons: the mechanical factors inherent in the growth of the streptococci on or in the heart valves are such that no bactericidal agent can be effectively applied.

The opinion was expressed several years ago by Lewis and Grant (70), and by Libman (71) that non-hemolytic streptococci probably circulate occasionally in the blood stream of most individuals. The recent observations of Kugel and Epstein (72) substantiate this opinion. The important factor in the production of disease rests not alone in the presence of micro-organisms in the blood stream, but in the ability of that particular micro-organism to elaborate substances which can set up certain types of tissue reaction, and also in the capacity of the tissues to respond in a particular manner to these substances. In the differences in the response of the tissues we see a possible explanation between the normal person, the man sick with rheumatic fever, and the individual with subacute bacterial endocarditis.

Syphilis.

A brief discussion of syphilitic heart disease is required not only because of the importance of the rôle of the treponema pallidum in the production of cardiac disorders, but also because the disease represents a condition where allergy has been shown to play a prominent part in the pathological changes encountered during the course of the infection. It has been demonstrated that without the development of such an allergic state the spirochetes may be tolerated by the body without exerting a harmful effect (73). They can lie dormant in tissues without these tissues showing any evidence of their presence until the general reactivity of the body is altered; then cellular changes in their neighborhood make evident the effort of the body to rid itself of these foreign invaders. There are also periods of relative immunity in contrast to hypersensitiveness on the part of the tissues, as is indicated by the work of Chesney and Kemp (74), Nichols (75) and others. But with the passage of years there develops an increasing degree of hypersensitiveness so that in an individual where early the general evidence of the reaction of the tissues is only a superficial macule or maculo-papule, later the response is many times as intense in the form of a nodule or gumma. I do not mean to imply that the late reactions always take the form of gummas, but rather that these lesions represent a relatively intense

form of tissue response to the presence of spirochetes. This disease also represents, par excellence, the importance of the rôle of lesions of the blood vessels in inducing alterations in the tissues supplied by them.

An appreciation of the nature of the evolution of syphilis is necessary if we are to find any reasonable explanation of the peculiarity of the cardiac symptoms. Although Brooks (76) and others have directed attention to the frequency with which evidence of cardiac involvement may occur early in the disease, it is clear from statistical studies, such as those made by Wyckoff and Lingg (77) as well as from the experience of clinicians and pathologists for decades, that the baneful effects of syphilis on the heart and aorta are not made very evident until years after the initial lesion. The occurrence of spirochetes in the blood early in the disease has been repeatedly demonstrated; hence we must conclude that the heart is exposed to them as much as are the other organs. But it is only after the passage of years accompanied by an ever-increasing degree of hypersensitiveness that the intensity of cardiac tissue response is sufficient to make a marked impairment of myocardial function clinically evident. There can be little doubt that during this period of so-called latency there may have been slowly progressing injury of the muscle; often lesions of the aorta and aortic valve have resulted in a compensatory hypertrophy of the myocardium, but it is only when this compensation fails or when progressive injury to the muscle fibers reaches a certain degree, or when lesions of the cardiac blood vessels are extensive enough to interfere seriously with an efficient blood supply to the heart muscle, that this muscle fails to work effectively. Warthin (78) has clearly shown how few spirochetes may be demonstrable in the sites of diffuse interstitial myocardial lesions, and that such lesions are not necessarily in the form of gummas. It is possible that just as gummas represent the most perfectly developed and typical form of marked allergic tissue response to treponema pallidum, so the Aschoff body may only represent a typical reaction to the causative agent of rheumatic fever. In both diseases there are diffuse types of inflammatory reaction and in both involvement of the blood vessels plays an important rôle in compromising the nutrition of the heart. In the manner in which the aorta and the aortic valves are involved the two diseases further resemble one another. A great difference in the two, however, is the rapidity and intensity with which involvement may take place in rheumatic fever and the slow-

ness with which it does occur in syphilis; nevertheless, chronicity and periods of activity alternating with intervals of apparent latency are often encountered in both maladies.

Septicopyemia.

The importance of septicopyemic involvement of the endocardium consists in the ease with which infected emboli are constantly swept into the blood stream and thus become carriers of the bacteria to other parts of the body. There is also the additional picture of severe intoxication and cachexia which is quickly reflected in a weakening of the poisoned heart muscle. These are both more deleterious factors in determining the outcome of the disease than is the ulceration of the valvular tissue resulting from the growth of the bacteria in the endocardium. In most instances a fatal issue occurs before the mechanical factor of inefficient heart valve, *per se,* can make itself felt.

Toxic Manifestations.

It is often difficult to determine just how great an influence a mild poisoning of the myocardium is exerting in the abnormal action of the heart encountered in most infectious diseases. The experiments of Newburgh and Porter (79) indicate that the heart may develop a type of resistance to the poisoning that results in Friedlander bacillus pneumonia, for when blood from infected animals was supplied to a normal animal's heart it quickly showed evidence of poisoning, whereas an infected dog's heart tolerated such blood much better. Levy (80) demonstrated that in patients with lobar pneumonia there was usually roentgenographic evidence of cardiac dilatation of from ten to thirty per cent. Whether this is a direct result of toxic action of some hypothetical poison on the myocardium, or a response of the heart to extra demands resulting from the general infection, or a combination of both factors, is a question not as yet decided. That such hearts will show the usual response to such cardiac tonics as digitalis was clearly demonstrated by the work of Cohn and Jamieson (81). Indeed, it may be stated as a general proposition that when in any infection, be it general or cardiac, the heart muscle and conduction system are too badly damaged to respond to digitalis, the patient is, indeed, in a precarious condition.

Conclusion.

As indicated early in this communication, no attempt has been made to cover completely all of the problems presented by the heart in infection. Indeed, to try to do this would prove futile, for the subject is one that could not be covered in its entirety in a large volume. An effort has been made rather to draw an outline in which certain well-recognized types of response of the respective cardiac tissues would provide a satisfactory explanation for the various features of heart disease in so far as active infection is concerned. I do not wish to leave the impression that any single type of cardiac infection includes only one type of tissue change; indeed, such is far from the case. The tissues in general have only a few ways in which they can respond to injury; the peculiar picture in any specific disease is determined by the relative amount of response of one or another type and also by the manner in which the special functions of any given organ or tissue are influenced by the particular structural changes.

In some instances an explanation of the nature of the structural changes is based on theories well substantiated by clinical and experimental evidence; in other instances on theories in the process of evolution. All that one can hope to accomplish in the way of a general description of such a complicated subject is to present a picture in which the various phenomena are correlated in a manner most in accordance with current knowledge and belief. With these reservations in mind we may conclude with a review of the type lesions of the heart in infection as being first toxic, as in diphtheria and early in scarlet fever; second allergic, hyperergic or hypersensitive, as late in scarlet fever, in rheumatic fever and in syphilis; third, bacteremic and biologically immune, as in subacute bacterial endocarditis; and fourth, bacteremic, toxic and cachectic, as in acute septicopyemia.

References.

1. Warthin, A. S.: The Myocardial Lesions of Diphtheria. J. Infect. Dis., 1924, xxxv, 32.
2. Baldasarri, V.: Beitrag zur Histologie der Endocarditis. Centralbl. f. allg. Path. u. path. Anat., 1909, xx, 97.
3. Czirer, L.: Über die Veränderungen an den Herzklappen bei akuten infektionskrankheiten. Virchows Arch. f. path. Anat., 1913, ccxiii, 272.
4. Jones, T. D., and White, P. D.: The Heart After Severe Diphtheria. Am. Heart J., 1927-1928, iii, 190.

5. Edmunds, C. W., and Cooper, R. G.: Action of Cardiac Stimulants in Circulatory Failure due to Diphtheria. J. A. M. A., 1925, lxxxv, 1798.
6. Escherich, Th., and Schick, B.: Scharlach. Vienna, 1912.
7. Nicoll, M.: Scarlet Fever. Nelson Loose Leaf Medicine, 1920, i, 451-468. T. Nelson & Son, New York.
8. Zinsser, H., and Grinnell, F. B.: Allergic Reactions to the Haemolytic Streptococcus. J. Immunol., 1925, x, 725.
9. Mackie, T. J., and McLachlan, D. G. S.: Experimental Sensitization to Culture Filtrates of Streptococcus Scarlatinae. Brit. J. Exper. Path., 1927, viii, 129.
10. Dochez, A. R., and Stevens, F. A.: Studies on the Biology of Streptococcus; Allergic Reactions with Strains from Erysipelas. J. Exper. Med., 1927, xlvi, 487.
11. Hector, F. J.: Effects of Scarlet Fever on Rheumatic Subjects. Arch. Dis. in Childhood, 1926, i, 339.
12. Romberg, E.: Über die Erkrankungen des Herzmuskels bei Typhus abdominalis, Scharlach und Diphtherie. Arb. a. d. med. Klin. zu Leipzig, 1893, 96.
13. Schmorl: Cited by Fahr, infra.
14. Fahr, Th.: Zur Frage der Herz—u. Gelenkveranderungen bei Gelenkrheumatismus u. Scharlach. Virchows Arch. f. path. Anat., 1921, ccxxxii, 135.
15. Aschoff, L.: Zur Myocarditisfrage. Verhandl. d. deutsch. path. Gesellch. 1904, viii, 46.
16. Geipel, P.: Untersuchungen über rheumatische Myokarditis. Deutsch. Arch. f. klin. Med., 1905, lxxxv, 75.
17. Curschmann, H., et al.: Anatomische, experimentelle und klinische Beiträge zur Pathologie des Kreislaufs. Arb. a. d. med. Klin. zu Leipzig, 1893.
18. Aschoff, L., and Tawara, S.: Die heutige Lehre von den pathologisch-anatomischen Grundlagen der Herzschwäche. Jena, 1906.
19. Poynton, F. J., and Paine, A.: Researches on Rheumatism. London, 1913.
20. Schottmüller, H.: Endocarditis lenta. München. med. Wchnschr., 1910, lvii, 1 and 617.
21. Libman, E., and Celler, H. L.: The Etiology of Subacute Infective Endocarditis. Am. J. Med. Sc., 1910, cxl, 516.
22. Swift, H. F.: Rheumatic Fever. Am. J. Med. Sc., 1925, clxx, 631.
23. Bezançon, F., and Weil, M. P.: La spécificité de la maladie rheumatismale. Ann. de méd., 1926, xix, 81.
24. Andrewes, C. H., Derick, C. L., and Swift, H. F.: The Skin Response of Rabbits to Non-Hemolytic Streptococci, I. Description of a Secondary Reaction Occurring Locally after Intradermal Inoculation. J. Exper. Med., 1926, xliv, 35.
25. Derick, C. L., and Andrewes, C. H.: The Skin Response of Rabbits to Non-Hemolytic Streptococci. II. Attempts to Determine Whether the Secondary Reaction is of the Nature of an Arthus Phenomenon. J. Exper. Med., 1926, xliv, 55.

26. Derick, C. L., and Swift, H. F.: Hyperergic Tissue Response to Non-Hemolytic Streptococci. Proc. Soc. Exper. Biol. and Med., 1927, xxv, 222.
27. Swift, H. F., and Derick, C. L.: Immune Tissue Response to Non-Hemolytic Streptococci. Proc. Soc. Exper. Biol. and Med., 1927, xxv, 224.
28. Swift, H. F., Hitchcock, C. H., and Derick, C. L.: General Tuberculin-like Reactions in Rheumatic Fever Patients Following Intravenous Injection of Streptococcus Vaccines or Nucleoproteins. Proc. Soc. Exper. Biol. and Med., 1928, xxv, 312.
29. Swift, H. F., Derick, C. L., and Hitchcock, C. H.: Bacterial Allergy (Hyperergy) to Nonhemolytic Streptococci in its Relation to Rheumatic Fever. J. A. M. A., 1928, xc, 906.
30. Swift, H. F., Derick, C. L., and Hitchcock, C. H.: Rheumatic Fever as a Manifestation of Hypersensitiveness (Allergy or Hyperergy) to Streptococci. Trans. Assoc. Am. Phys., 1928, xliii, 192.
31. Kuczynski and Wolff: Beitrag zur Pathologie der Experimentellen Streptokokkeninfektion der Maus. Verhandl. d. deutsch. path. Gesellsch., 1921, xviii, 47.
32. Tillett, W. S.: Studies on Immunity to Pneumococcus Mucosus (Type III). III. Increased Resistance to Type III Infection Induced in Rabbits by Immunization with R and S Forms of Pneumococcus. J. Exper. Med., 1927, xlvi, 343; and personal communication.
33. Birkhaug, K. E.: Rheumatic Fever. J. Infec. Dis., 1927, xl, 549.
34. Kaiser, A. D.: Skin Reactions in Rheumatic Fever. J. Infect. Dis., 1928, xlii, 24.
35. Small, J. C.: The Bacterium Causing Rheumatic Fever and a Preliminary Account of the Therapeutic Action of Its Specific Antiserum. Am. J. Med. Sc., 1927, clxxiii, 101.
36. Small, J. C.: Observations Bearing on the Specificity of Streptococcus Cardioarthritidis in Rheumatic Fever and Sydenham's Chorea. Am. J. Med. Sc., 1928, clxxv, 638.
37. Small, J. C..: The Present Development of the Biologic Products of Streptococcus Cardioarthritidis and Their Application in the Treatment of Rheumatic Diseases. Am. J. Med. Sc., 1928, clxxv, 650.
38. Strauss, H.: Allergische Phänomene bei rheumatischen Erkrankungen. Deutsch. med. Wchnschr., 1927, liii, 737.
39. Mackenzie, G. M., and Hanger, F. M.: Allergic Reactions to Streptococcus Antigens. J. Immunol., 1927, xiii, 41.
40. Zinsser, H.: On the Significance of Bacterial Allergy in Infectious Diseases. Bull. New York Acad. Med., 1928, iv, 351.
41. Von Glahn, W. C.: Rheumatic Diseases of the Heart. Arch. Path. and Lab. Med., 1927, iii, 355.
42. Von Glahn, W. C.: Auricular Endocarditis of Rheumatic Origin. Am. J. Path., 1926, ii, 1.
43. Pappenheimer, A. M., and Von Glahn, W. C.: Lesions of the Aorta Associated with Acute Rheumatic Fever, and with Chronic Cardiac Disease of Rheumatic Origin. J. Med. Res., 1924, xliv, 489.

44. MacCallum, W. G.: Rheumatism. (The Harrington Lecture.) J. A. M. A., 1925, lxxxiv, 1545.
45. Swift, H. F.: The Pathogenesis of Rheumatic Fever. J. Exper. Med., 1924, xxxix, 497.
46. Levy, R. L., and Turner, K. B.: Variations in Auriculoventricular Conduction Time in Rheumatic Carditis with Salicylate Therapy. Proc. Soc. Exper. Biol. and Med., 1927, xxv, 64.
47. Swift, H. F., Derick, C. L., and Andrewes, C. H.: Valvulitis Early in Rheumatic Fever. (In press.)
48. Coombs, C. F.: Rheumatic Heart Disease. Bristol, 1924.
49. Siegmund, H.: Über einige Reaktionen der Gefässwände und des Endokards bei experimentellen und menschlichen Allemeininfektionen. Verhandl. d. deutsch. path. Gesellsch., 1925, xx, 260.
50. Dietrich, A.: Versuche über Herzklappenentzündung. Ztschr. f. d. gesamte exper. Med., 1926, l, 85.
51. Kugel, M. A., and Gross, L.: Gross and Microscopic Anatomy of the Blood Vessels in the Valves of the Human Heart. Am. Heart J., 1925, i, 304.
52. Winternitz, M. C.: Personal communication. Woodruff, C. E.: Studies on the Vasa Vasorum. Am. J. Path., 1926, ii, 567.
53. Kerr, W. J., and Mettier, S. R.: The Circulation of the Heart Valves. Am. Heart J., 1925, i, 96.
54. Bayne-Jones, S.: The Blood Vessels of the Heart Valves. Am. J. Anat., 1917, xxi, 449.
55. Von Glahn, W. C., and Pappenheimer, A. M.: Specific Lesions of Peripheral Blood Vessels in Rheumatism. Am. J. Path., 1926, ii, 235.
56. Pappenheimer, A. M., and Von Glahn, W. C.: Studies in the Pathology of Rheumatic Fever; Two Cases Presenting Unusual Cardiovascular Lesins. Am. J. Path., 1927, iii, 583.
57. Kugel, M. A., and Epstein, E. Z.: Proc. New York Path. Soc., April, 1928 (not yet published), and Arch. Path., 1928, vi, 247.
58. Rosenow, E. C.: Experimental Infectious Endocarditis. J. Infect. Dis., 1912, xi, 210.
59. Kinsella, R. H.: Bacteriologic Studies in Subacute Streptococcus Endocarditis. Arch. Int. Med., 1917, xix, 367.
60. Libman, E.: Observations on Subacute Streptococcus (and Influenzal) Endocarditis. Brit. Med. J., 1920, ii, 304.
61. Kinsella, R. A., and Sherburne, C. C.: Experimental Production of Streptococcus Endocarditis. J. A. M. A., 1923, lxxx, 1643.
62. Wright, H. D.: The Production of Experimental Endocarditis with Pneumococci and Streptococci in Immunized Animals. J. Path. and Bact., 1926, xxix, 5.
63. Wadsworth, A. B.: A Study of the Endocardial Lesions Developing During Pneumococcus Infection in Horses. J. Med. Res., 1919, xxxix, 279.
64. White, Benjamin: Personal communication.

65. Clawson, B. J.: Studies on the Etiology of Acute Rheumatic Fever. J. Infect. Dis., 1925, xxxvi, 444.
66. Howells, K. M., and Corrigan, M.: Skin Reactions with Bacterial Filtrates of Anhemolytic Streptococcus, Hemolytic Streptococcus, and B. Typhosus. J. Infect. Dis., 1928, xlii, 149.
67. Kinsella, R. A., and Garcia, O.: A Clinical Experiment in Subacute Streptococcus Endocarditis. Proc. Soc. Exper Biol. and Med., 1925, xxiii, 136.
68. Rothschild, M. A., Sachs, B., and Libman, E.: The Disturbances of the Cardiac Mechanism in Subacute Bacterial Endocarditis and Rheumatic Fever. Am. Heart J., 1927, ii, 356.
69. Cohn, A. E., and Swift, H. F.: Electrocardiographic Evidence of Myocardial Involvement in Rheumatic Fever. J. Exper. Med., 1924, xxxix, 1.
70. Lewis, T., and Grant, R. H.: Observations on Subacute Infective Endocarditis. Heart, 1923, x, 21.
71. Libman, E.: Infections microbiennes généralisées. La Presse Méd., 1924, xxxii, 881.
72. Kugel, M. A., and Epstein, E. Z.: Proc. New York Path. Soc., 1928, April (not yet published), and J. Infect. Dis., 1929 (in press).
73. Levaditi, C., and Yamanouchi, T.: Recherches sur l'incubation dans la syphilis. Ann. de l'Inst. Pasteur, 1908, xxii, 763.
74. Chesney, A. M., and Kemp, J. E.: Further Observations on the Possibility of Cure of Syphilis in the Rabbit with Arsphenamine. J. Exper. Med., 1925, xlii, 17.
75. Nichols, H. J.: Experimental Immunity in Syphilis and Yaws. Am. J. Trop. Med., 1925, v, 429.
76. Brooks, H.: The Heart in Syphilis. Am. J. Med. Soc., 1913, cxlvi, 513.
77. Wyckoff, J., and Lingg, C.: Etiology in Organic Heart Disease. Am. Heart J., 1926, i, 447.
78. Warthin, A. S.: The New Pathology of Syphilis. Am. J. of Syphilis, 1918, ii, 425.
79. Newburgh, L. H., and Porter, W. T.: The Heart Muscle in Pneumonia. J. Exper. Med., 1915, xxii, 123.
80. Levy, R. L.: The Size of the Heart in Pneumonia. Arch. Int. Med., 1923, xxxii, 359.
81. Cohn, A. E., and Jamieson, R. A.: The Action of Digitalis in Pneumonia. J. Exper. Med., 1917, xxv, 65.

THE EFFECT OF STRAIN ON THE HEART.

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The effect of strain on the heart has been a subject of great interest and importance for generations but we are to-day in a better position to discuss it than has been possible before. From laboratory experiments and from clinical investigations have come constantly in recent years new observations which give us a clearer idea of the influence of various factors on the circulation, though difficult problems still remain unsolved. The arteries, capillaries and veins, muscular movements, respiration and gravity all have important functions to perform in maintaining the circulation and in aiding in its adjustment to various conditions, but the heart must continue to be regarded as the chief mechanism in the distribution of blood throughout the body, and as we shall see, it must be viewed as a double organ with the effect of strain sometimes on both sides, but often preponderantly on one side only.

The effect of strain on the heart concerns not only the experimental pathologist, the physiologist, the general practitioner and the specialist in cardiovascular disease, but it concerns you all. We must know not only the effect of strain on the normal heart but also the effect of strain on the diseased heart. The subject is one of large scope as we must realize that there are not only various kinds of strain and that each kind of strain may vary in degree, but that the heart itself may vary in its strength, reserve power, and degree of health. Finally there may act on the same heart simultaneously various kinds and degrees of strain. With so many variables, it is obvious that but general statements can be made, and these must be applied to each individual case with great care. Here the experience and wisdom of the clinician in the practice of medicine are of utmost importance in the weighing of evidence which will determine the diagnosis, prognosis and treatment of disturbances of the circulation. I can here but outline briefly the effect on the heart of strain in general and that of various individual factors. The complex assembly of individual factors in special cases must be passed over with mention merely of some of the more frequent groupings.

The direct effects of strain on the heart vary essentially with the kind and degree of the strain and the condition of the heart to start with. Slight strain may affect a diseased heart even more than marked strain a normal heart. The constant repetition of strain, that is chronic strain, may be completely taken care of by the cardiac reserve; if it is not, the myocardium must meet the strain by increasing in strength. Its tone first is improved, then comes actual increase in bulk with enlarged muscle fibers and probable increase in capillary circulation. Whether general cardiac dilatation and muscle injury must always precede actual hypertrophy is not yet clear; sometimes at least this appears to be true. Then with increasing or persistent strain and decreasing reserve the heart begins to fail. If the strain is on both ventricles the signs of failure may indicate this—congestion of lungs, liver and neck veins, dependent oedema, and finally general anasarca with orthopnea and cyanosis. But where the strain is largely unilateral then the hypertrophy and dilatation may be largely limited to the right or to the left ventricle and failure may start in that ventricle, the other maintaining its work, perhaps too effectively for a while, until it fails also. In aortic valve disease, chronic hypertension and often coronary disease, especially where there is thrombosis of the descending branch of the left coronary artery, the left ventricle hypertrophies and dilates, with little change in the right ventricle. Then comes failure with dyspnea and pulmonary oedema and general slowing of peripheral circulation with oedema of the feet. If the failure is acute we may have in such cases cardiac asthma, probably due largely to the inability of the left ventricle to take care of the blood being sent to it by the more vigorous right ventricle. In mitral stenosis on the other hand or in infarction limited to the right ventricle the immediate effects are different. The right ventricle is hypertrophied and dilated. Its failure is shown in large part by its inability to take care of the blood which it receives. Engorgement of neck veins and liver, ascites and finally general anasarca develop. The pulmonary circulation also is slowed. The preponderant failure of one ventricle does not generally last very long, the other ventricle also fails or the first ventricle recovers. When right ventricular failure is added to left ventricular failure, some of the dyspnea and pulmonary oedema may actually decrease but signs of right ventricular failure are added. The balance is a delicate one and all gradations and relative degrees of failure of either ventricle or both may occur. Dr. G. Canby Robinson has

recently called our attention anew to the importance of this ventricular balance.

Acute strain may be and usually is well taken care of by the cardiac reserve but if the heart is weak or the insult a severe one, as in coronary thrombosis, acute dilatation of the heart may occur. This is less common than the use of the term itself which has been applied wrongly to all sorts of conditions such as paroxysmal tachycardia and syncope.

Heart strain may be *intrinsic or extrinsic in origin* and I propose to consider first the intrinsic factors which are what we generally regard as structural defects, or lesions, and disorders of rhythm. Frequently there is a combination of disordered heart beat and diseased valve or muscle, which results in incapacity and death where neither one alone might be dangerous.

A. *Intrinsic Strain.*

1. Let us consider first *valvular disease* as a condition giving rise to heart strain. Years ago valvular regurgitation and stenosis were the chief topics discussed under the subject of heart disease, then in recent times the pendulum swung far to the other extreme and the condition of the myocardium was regarded as of paramount importance and fully able to compensate for the valvular defects. Now we appreciate that no matter how good the heart muscle may be it may fail under the burden of long continued marked valvular disease. We must view with considerable respect a high degree of mitral stenosis or of aortic regurgitation; aortic stenosis and mitral regurgitation are less serious. Slight valvular involvement is compatible with long life and full physical and mental activity, but even in such a case the heart usually shows some enlargement. Generally speaking, the greater the degree of either regurgitation or stenosis of any valve the greater is the strain on the heart, often unevenly distributed to one or the other ventricle; the greater this strain the shorter the life. Given aortic regurgitation for example, if the defect is a slight one there may be no symptoms whatsoever through a strenuous long life, the heart may be but very slightly enlarged and the blood pressure normal. If the defect is of high degree the heart becomes enormous, the pulse pressure extensive and life is usually shortened, no matter what the condition of the muscle, though of course a healthy myocardium will hold out longer than one that is

diseased. How to keep the myocardium healthy in such cases is often a problem. Avoidance of infections and of undue fatigue is of the greatest importance. The heart is already exercising energetically where the valves are deficient and one is apt to conclude that no extra bodily exercise other than the routine quiet daily life should therefore be allowed. But it is probable that some exercise, if not severe and if well tolerated, helps the heart by improving the tone of the circulation in general. A state of physical fitness and of good physical training is undoubtedly an asset and if the heart can stand a slight to moderate amount of healthy exercise it should in turn be benefited. Much judgment must be applied in individual cases.

a. *Mitral disease* acts in various ways as a strain on the heart. If *regurgitation* is preponderant, and such a condition does exist, there is extra work for the left ventricle, as in aortic regurgitation, and for the left auricle and the right ventricle. The strain results eventually, if the mitral regurgitation is of sufficient degree, in hypertrophy of the whole heart with dilatation of the left ventricle and left auricle. The left ventricle has to hold enough blood to make up for the amount regurgitated into the left auricle, and to increase in muscle mass sufficiently to take care of this increased work of pumping blood in two directions. The left auricle tends to be dilated by the blood regurgitated into it during ventricular systole, and the right ventricle increases in strength and in muscle mass to overcome the back pressure from the left ventricle. Eventually as the heart fails the right auricle also may dilate but it is not primarily affected. With chronic marked mitral regurgitation either ventricle may fail from the strain, or both together. It is one of the few conditions which act as an immediate strain on both sides of the heart.

With the development of functional mitral regurgitation due to dilatation of the mitral ring in failure or dilatation of the left ventricle as in chronic hypertension, coronary thrombosis or aortic regurgitation, the right ventricle too is put under a strain where previously only the left ventricle had been affected. The right ventricle then enlarges and in its turn may fail.

Mitral stenosis puts a strain on the left auricle and right ventricle, both of which frequently become very large and fail, usually with the appearance of auricular fibrillation. The left ventricle may remain unaffected or even seem smaller than normal with the

decreased work. With the decreased oxygenation of the blood in the lungs cyanosis appears; this is probably due to the insufficient exposure of the venous blood to the alveolar air because of the swelling of the pulmonary blood vessels. The pulmonary pressure is raised and there may be pulmonary apoplexy. When the right ventricle fails the pulmonary circulation becomes slowed, there is more stasis there because of the drop in pressure, but the chief signs of the failure appear in the form of engorgement of the veins of neck and arms, congestion of the liver and finally peripheral oedema and increased cyanosis.

It must be remembered that all grades of mitral regurgitation and mitral stenosis exist with combinations of the two. An attempt has been made by surgery to convert marked mitral stenosis into a certain degree of mitral regurgitation, as yet unsuccessfully. It is likely that mitral stenosis and regurgitation combined, each of moderate degree, exert a less serious strain on the heart than either condition alone, if of high degree.

b. *Aortic valve disease* acts primarily as a strain only on the left ventricle, whether there is stenosis or regurgitation. The left ventricle hypertrophies and dilates, but only as it fails and mitral regurgitation develops is there a direct effect on the other chambers of the heart, though there is an indirect and probably very important deleterious effect on the coronary circulation in cases of marked aortic regurgitation. Aortic stenosis is a less serious lesion than aortic regurgitation except when the regurgitation is of but slight degree. It is found not rarely in old people with fairly competent hearts. I regard it as a favorable complication of aortic regurgitation. With aortic stenosis alone clinically or with aortic stenosis and regurgitation combined the heart is less enlarged than with aortic regurgitation without stenosis. This may be due in part to the bad effect on the coronary circulation of marked aortic regurgitation. Physical examination, X-ray study and electrocardiography all reveal the enlarged left ventricle found in many cases of chronic aortic valve disease, just as these same methods show the large right ventricle in well-marked mitral stenosis. When the left ventricle fails the peripheral circulation is decreased, but the first definite evidence usually comes in the form of dyspnea and pulmonary oedema, and sometimes acute attacks of cardiac asthma, probably due primarily as already stated to congestion of the pulmonary circulation because of the inability of the left ventricle to dispose of the blood passed on to it by the

more normal right ventricle. With recovery of the left ventricle by rest, orthopnea, bleeding, morphine or digitalis therapy the pulmonary oedema and orthopnea disappear, or the same thing may happen temporarily when the right ventricle fails in its turn and blood accumulates in the liver, and oedema peripherally, instead of in the lungs.

Aortic regurgitation associated with luetic aortitis is generally much more of a strain on the heart than that due to rheumatic endocarditis. The lesion is apt to be more extensive, it is more progressive, and there is often associated coronary orifice involvement in the aortitis with still greater diminution of coronary circulation. Almost the largest heart known is found with marked aortic regurgitation—the cor bovinum, the size indicating in this instance at least the importance of the strain from the valvular lesion.

c. *Tricuspid valve disease* of any great moment is very rare and may be dismissed with the observation that it acts on the right side of the heart, whether stenotic or regurgitant in type, much as does mitral disease on the left side. When it is present it is usually overshadowed by the much more important and marked mitral disease.

d. *Pulmonic valve disease* is also uncommon; as an acquired defect it is very rare and whether regurgitant or stenotic in type acts on the right ventricle much as aortic valve disease acts on the left ventricle. Congenital pulmonic stenosis if of high degree is very serious, is usually associated with marked enlargement of the right ventricle, often with associated congenital cardiac defects and with cyanosis and a short life. The cyanosis in such cases is due primarily to the right to left shunt of unoxygenated blood through a septal defect and not to the pulmonic stenosis itself.

2. *Congenital defects of the heart* vary very much in the strain they cause. Some are of little or no significance, like small septal defects or patency of the ductus arteriosus, to be found only on routine physical examination; others are so complex and serious that they allow survival for only a few minutes, hours or days after birth. There are all degrees of strain represented. In general it may be said that the degree of strain in the victim varies in proportion to the cyanosis. Circulatory failure and death in such cases frequently are precipitated by infections, like pneumonia.

3. *Pericarditis* sometimes acts as a great burden for the heart and sometimes it too is negligible. There may be a transient unimportant pericardial friction rub leaving perhaps a milk spot, or there may be

an extensive pericardial effusion endangering life by its rapid accumulation with pressure on the heart and great vessels. Paracentesis here may save lives but usually the degree of effusion is such that tapping is not necessary. The orthopneic position with great distress particularly in breathing, and a marked paradoxical pulse are often evidence, aside from thoracic signs, of the presence of a massive effusion. The speed of accumulation makes a great difference—a slowly gathering tubercular effusion may be well supported even when enormous.

Chronic pericarditis may also vary in its effect on the heart dependent on its degree and location. If the heart is adherent to diaphragm, ribs or mediastinum and at each contraction must pull on these structures it is under great strain and may become very large, mainly due to hypertrophy of the left ventricle but sometimes of the whole heart. Some of the largest hearts of all are found with chronic mediastinopericarditis, 1,000 grams or more in weight. There may be chronic obstruction and stasis with engorgement of the liver giving rise to Pick's Disease. So-called cardiolysis—a removal of the ribs to which the heart is attached—has in rare cases afforded relief to the overworked heart. Frequently chronic adhesive pericarditis is so slight in degree that there is no heart strain at all and no enlargement, and the condition therefore may not be diagnosable during life.

4. *Coronary disease* whether arteriosclerotic or infectious in nature acts as a strain on the heart by cutting down the blood supply. The greater the degree, the greater the strain. Coronary anastomoses and the efficiency of the Thebesian circulation may vary very much in different individuals and the same degree of coronary disease may thus act very differently in different people. Generally actual coronary thrombosis is a serious burden; it may result in hypertrophy as well as dilatation, the latter apparently coming first, which would seem to substantiate recent opinion that injury to heart muscle may precede hypertrophy. Whether lesser grades of coronary disease may also cause increase in heart weight is still open to question—it would seem possible that increased demand on that part of the myocardium which still functions well may cause it to increase in bulk. The strain of coronary disease may be on either ventricle but is more marked usually on the left, especially when there is actual coronary thrombosis. The left ventricle may fail suddenly or slowly.

5. *Myocardial disease,* not due to coronary involvement, but *infectious* or *toxic* in nature, may place a very great burden on the heart. An acute myocarditis as in diphtheria or rheumatic fever may precipitate failure and death. It is likely that a few chronically damaged enlarged hearts have resulted from real myocarditis, usually of unknown but sometimes of luetic origin.

6. *Disease of the great vessels* although strictly extracardiac may best be included here. There is so far as we know no especial strain on the heart from lesions of these great vessels themselves if the heart is not simultaneously involved except under two circumstances. Stiffening of the aortic wall with loss of elasticity does doubtless increase the work of the heart by reducing the coöperation of the aorta in maintaining a satisfactory circulation. The strain from this is probably not very great however and is readily taken care of; usually the heart does not hypertrophy. Arteriovenous aneurysms and patency of the ductus arteriosus if extensive, do very materially increase the work of the heart and cause left ventricular enlargement somewhat like that caused by aortic or mitral regurgitation. Correction of the arteriovenous aneurysm causes the heart to decrease in size. Ordinary aortic aneurysms or aortitis do not affect the heart appreciably unless the coronary orifices are involved or aortic valve deformed.

7. *Disturbances of cardiac rhythm* may sometimes, if marked, cause cardiac strain. Premature contractions, or extrasystoles, whether auricular or ventricular in origin, rarely if ever, are any kind of a burden for the heart. If they occur so often (every other beat or more frequently) as to interfere with the efficiency of the circulation the myocardium too may be slightly affected but such a condition is very uncommon. Paroxysmal tachycardia, auricular flutter and auricular fibrillation on the other hand do, through the speed of the heart, impose a burden on both ventricles which may result in heart failure if very long continued at a very rapid heart rate, especially if there are other factors like valvular or coronary disease or hypertension. Flutter and fibrillation are more important than paroxysmal tachycardia except that of ventricular origin which is generally associated with serious heart disease. Heart block is a strain for the heart only in reducing the coronary circulation if the pulse becomes extremely slow. Rare disturbances of rhythm like auriculo-ventricular nodal rhythm or sinoauricular block are of little moment.

B. *Extrinsic Factors.*

Extrinsic factors causing heart strain are of far less importance than the intrinsic factors already discussed with two exceptions. They are hypertension and hyperthyroidism.

1. *Hypertension,* either with or without renal disease, is a great burden for the heart if the blood pressure remains high for a long time. The higher the pressure and the longer it is maintained the greater the strain. The effect is primarily on the left ventricle with enlargement, found usually by physical examination, X-ray and electrocardiogram. Eventually the left ventricle may fail and the symptoms and signs of this failure already mentioned may appear. The course may be shortened by the coincident effect of coronary disease, uremia or other complication, but a myocardium apparently in good condition and enormously hypertrophied may fail because of the chronic burden of the hypertension alone. The higher the diastolic pressure the worse the prognosis. The cause of essential hypertension is still open to question but apparently it is due to increased peripheral vascular resistance. Bazett makes the interesting observation that "If the minute volume of the heart be kept constant, and a 12 per cent diminution in length of fibers occurred in all of the arterioles since these offer the main part of the resistance, the mean pressure would have to be almost doubled."

2. *Hyperthyroidism* may be a cause of important heart disease and failure unless diagnosed and treated early. Even lesser grades undoubtedly increase the work of the heart but the severe grades impose a great burden on the circulation, with tachycardia and increased pulse pressure associated with the increased metabolic rate. It appears doubtful if any actual toxic myocarditis occurs and the ultimate effect may be simply that of cardiac muscle exhaustion. Operation if properly done generally puts an end to this heart trouble.

3. *Exercise and work.* That very vigorous exercise and work may prove too great a strain for a diseased or untrained heart is unquestioned but under proper conditions the most strenuous exertion is well borne by a healthy heart. There is still somewhat of a doubt about the possibility of the heart's enlarging as the result of exercise. Abnormal hearts undoubtedly may enlarge—both acutely and chronically, but the normal heart probably increases only a little in size at most; its reserve is great. DeMar, the champion marathon runner, shows no cardiac enlargement by X-ray or physical examination, but

some observations on professional athletes abroad have indicated that there may be slight enlargement of the heart in professional cyclists, oarsmen and ski-racers. This seems reasonable from comparison with animal studies. The hearts of athletic animals are larger than those of non-athletic animals of the same size whether of the same species or different ones. For example, the heart of the hare is relatively three times as large as the heart of the rabbit. William Harvey, in his memorable Disquisition on the Motion of the Heart and Blood in Animals published just three hundred years ago, wrote as follows: "The more muscular and powerful men are, the firmer their flesh, the stronger, thicker, denser, and more fibrous their heart, in the same proportion are the auricles and arteries in all respects thicker, closer, and stronger." It is not likely that hypertension alone accounted for these observations of Harvey's. Exercise must be prescribed for people with heart disease with care but as already stated, if the heart is competent a little exercise is probably beneficial. The poor physical condition resulting from total abstinence from exercise is not conducive to the best of health. A man with aortic regurgitation but otherwise in good health and accustomed to some exercise is likely to be a better risk in a serious infection or operation than a lazy fat man with a normal heart.

The so-called *athletic heart,* as I have seen it, has consisted simply in effort syndrome or in an "irritable heart" in a nervous overtrained or undertrained person and not in heart disease.

The factor of long years of *hard physical work* in the production of coronary sclerosis is still an uncertain one. Senility, hypertension and syphilis probably played a part in some of the cases so designated years ago.

Military service has sometimes been blamed for the production of heart disease but practically all soldiers with heart symptoms and signs can be shown to belong to the familiar groups of effort syndrome or of organic heart disease due to rheumatic, luetic, hypertensive or arteriosclerotic factors. Rarely extreme strain in military service may perhaps do direct harm. The usual "soldier's heart" is of course simply the effort syndrome.

Tests to determine the efficiency of the heart, whether of the nature of exercise or of respiratory tests, concern physical fitness as a whole rather than cardiac fitness in particular.

It is of interest to note that in the quiet relaxed upright position the heart is under less strain than when the body assumes the recum-

bent position, due in large part to the difference in the amount of blood flow in these positions. Sufferers from cardiac asthma, angina pectoris decubitus, or simple orthopnea, appreciate this fact tremendously.

4. *Accidents* do not act as a special heart strain unless of very unusual nature affecting the heart directly by injury or by causing rare valve rupture. When heart disease is found after accidents it was almost invariably present before but not recognized or looked for. Accidents may precipitate cardiac symptoms, however, as a kind of "last straw" and to that extent may be considered responsible. For the accident insurance board it should be possible to grade the compensation according to the responsibility of the accident, as is the law in Kentucky, and not to be obliged, as in most states, to consider it wholly or not at all responsible.

Cardiac accidents and strain may readily result from *constipation* chiefly due to the effort, which may sometimes be very great, expended in defecation. Here the strain, which is essentially that of the Valsalva experiment, may precipitate angina pectoris or cardiac asthma in a diseased heart although a normal heart withstands the effort easily. Coitus also may cause angina pectoris or cardiac asthma or increase of congestive failure in a heart with little or no reserve.

5. *Pregnancy*. For the normal heart pregnancy is not a strain. Doubtless it does increase the work of the circulation, including that of the heart, and there may even be some temporary increase in heart size. Most of the apparent increase in the size of the heart in the later months of pregnancy is due, however, to its upward displacement by the abdominal tumor. Cardiac patients vary markedly in their ability to stand the strain of pregnancy and childbirth. In general, they go through it all right, but each case must be carefully judged by herself and followed conscientiously. Marked mitral stenosis, marked aortic regurgitation, marked cardiac enlargement and auricular fibrillation make termination of pregnancy advisable; congestive failure demands such a measure.

6. *Obesity* and *overeating* are important strains only for a diseased heart, but they are often associated with conditions like hypertension and coronary disease that cause heart damage. Much of the so-called fatty heart is a misnomer. Cardiac symptoms in an obese person may come from poor general physical condition with faulty

muscular tone throughout the body—really a type of effort syndrome—or else from associated organic heart disease. Probably actual encroachment or invasion of the heart by fat is of little moment except when it indicates a disease process. A hearty meal may precipitate angina pectoris and coronary thrombosis, and thus occasion the wrong diagnosis of "acute indigestion."

7. *Infectious diseases* although doubtless increasing the work of the heart somewhat during the height of fever and tachycardia and depressing the myocardium when there is much toxicity, do not exert any great particular strain on the normal heart unless they actually cause heart disease as in the case of serious rheumatic fever or diphtheria. The routine administration of digitalis therefore in infectious disease appears unwarranted although with the greater acute strain in pneumonia it is possible that it may do some good. This still remains open to question and even in pneumonia it may be wisest to withhold cardiac stimulation unless it is obviously needed, either as an emergency measure or in the treatment of known cardiac patients (and perhaps old people). Most failure in infectious disease is due to vasomotor and not to cardiac collapse.

8. *Disease in other organs* may cause strain to the heart, usually of but slight degree. Pulmonary disease by displacement of the heart or by increasing the work of the right ventricle, distention of the abdomen with gas or fluid and pressure upward on the heart, and nephritis by producing uremic intoxication are examples of disorders which may affect the heart. Most local diseases or focal infections, like cholecystitis, apical tooth abscess or prostatitis, act if at all on the heart only to irritate it. Premature beats or even paroxysmal tachycardia or angina pectoris may be precipitated temporarily, to clear up when the local disease is relieved. However angina pectoris and myocardial disease are not actually caused by such a condition. They are merely increased or brought to light, although it is possible that degenerative or inflammatory changes in the coronary arteries may be initiated or hastened by reaction to focal infection; that is still an open question. Hypertension and diabetes appear especially to favor the occurrence of angina pectoris.

9. *Anemia* whether secondary or primary is, if severe, quite a burden for the heart in that the myocardium is directly affected. Dilatation may result, murmurs appear, and angina pectoris be precipitated.

10. *Nutritional diseases,* beri-beri in particular, may cause myocardial weakness, degeneration and failure. It is possible that in slight degree they may frequently cause some trouble.

11. *High altitudes* exert a definite strain on the circulation, including the heart, if high enough to reduce the oxygen content of the air considerably. Some individuals can adjust themselves easily to high altitudes, others do so with difficulty, whether or not there is heart disease. It is likely that altitudes below 10,000 feet impose relatively little burden on the heart if healthy but for patients with heart failure low altitudes are desirable.

12. *Anaesthesia and surgical operations* exert a variable strain on the heart but less on the heart apparently than on the nervous control of the circulation. If anaesthesia is well given and operations are well performed both cardiac and non-cardiac patients stand the strain well. There are rare cardiac accidents, now and then paroxysms of tachycardia usually of little moment, and rarely failure; most trouble of circulatory type is of the nature of shock for which cardiac therapy is not necessary. It need hardly be said that in preparation for operation, digitalis is rarely required. Of the general anaesthetics at present ethylene seems to be particularly favorable from the standpoint of the circulation.

13. *Forced fluids.* The custom of the forcing of fluids much in vogue in therapy in late years is not always without its drawbacks, one of which is undue strain on the heart. This, if the heart is weak, may actually lead to congestive failure. That the ingestion of great quantities of fluid may actually lead to cardiac hypertrophy and failure is indicated by the occurrence of the *beer heart* in Europe.

14. *Alcohol, tobacco, coffee.* There is little evidence that these substances produce any deleterious effect on the heart except transiently as irritants, or through its toxic effect in the case of marked chronic alcoholism. It is, however, quite certain that disturbing disorders of cardiac rhythm like paroxysmal auricular fibrillation may actually follow overuse of alcohol, tobacco, or both combined. Excessive use of tobacco has in some cases precipitated or increased angina pectoris.

15. Finally, we come to the *effect of nervous* (mental and emotional) *strain* on the heart. Through nerve action this is undoubted, and in this present era when nervous strain is almost a continuous one for people in many communities it is doubtless more of a factor than ever before. Business, professional and social competition is

extremely active. The telephone and automobile are always exciting our nerves. The so-called effort syndrome, angina pectoris, and perhaps hypertension and presenile arteriosclerosis, appear to be increasing; nervous strain is probably in part responsible.

In addition to the factors of cardiac strain that I have discussed there are doubtless others not known or poorly understood, but I have presented enough to show the complicated situation which may be found in a given individual and the care that is needed in the analysis of cardiac strain. On the whole, however, the heart is an extraordinarily powerful organ resisting strains of all kinds with great ability.

COMMENTS ON THE TREATMENT OF HEART DISEASE.

BY JAMES B. HERRICK, M.D.,

Chicago.

In this address there might properly be considered the preventive treatment of heart disease. This would include the proper management of acute and chronic general and focal infections whose relation to acute carditis and so-called chronic fibrous myocarditis is rationally assumed, rather than proven, to be rather close. There would be stressed the prevention and early treatment of syphilis, a prolific cause of aortic and cardiac disease. Above all there would be emphasized the importance of doing what can be done, to guard against rheumatism. Coöperation in the international study of rheumatism would be urged as a privilege and a duty for American medicine. There would also be mention of regulation of modes of living, in the attempt to prevent or postpone the early breakdown of the heart of valvular or myocardial disease or of hypertension. So, too, there would be noted a group that might be called potential cardiacs, those who are inclined toward or "threatened" with heart disease and for whom advice as to right living may mean much. In this class might be put the obese, the gouty, the emphysematous, the kyphoscoliotic, the plethoric, the anemic and undernourished individual, psychoneurotics of certain types, and those with the stigma of hereditary early cardiovascular degeneration. And one would applaud the efforts now being put forth and under contemplation, to learn more of the essential nature and rational means of handling the degenerative processes of adult and senescent years concerning which there is so much speculation and so little accurate knowledge.

I shall, however, omit consideration of these matters of prophylaxis. Nor will I dwell upon the latest advances in cardiac therapy—surgical, dietetic, medicinal. Some of the writings concerning them are understandable; others seem merely a maze of words. Some of these newer therapeutic measures strike one as promising, others as far-fetched. All of them may better be considered in the quiet of the study or, if attractive, tried out in the laboratory or the hospital ward.

Therefore, departing a little from the custom, I shall call attention to certain undesirable or even harmful tendencies that are concerned rather with the art than with the science of medicine. Though not new, they seem worthy of rediscussion at this time when the attention

of physician and layman is so strongly focused on disease of the heart.

The cynic said to me, when he heard that I was to discuss the treatment of heart disease: "Why not condense and say there is no treatment for heart disease save rest and digitalis?" On being hard pressed he admitted that at times opium or a cathartic might be helpful. Really, the cynic is not far wrong. The great majority of our heart cases are treated along these lines. Rest to permit recovery of muscular tone and to prevent overstrain of a damaged organ. Digitalis to slow conductivity and heart rate and thus secure more rest, together with more vigorous ventricular contractions and more normal output of blood with each beat. Opium to relieve pain, induce sleep and lessen nerve strain. A laxative for purposes of elimination. This is good treatment and need not be discussed. But there are occasional misapplications of the general principles to which reference may properly be made.

Consider the subject of rest. How much during or after an acute carditis? How much following a cardiac breakdown or after a coronary occlusion? How much in the case of a fibrillating auricle with mitral stenosis or in the failing heart of hypertension, the patient in either case perhaps able to be up and about?

These are not easy questions to answer. Fixed rules cannot and should not be laid down. There seems to be a wide difference in the practice of different physicians. Some prescribe too little rest for acute inflammation of the heart and for serious breakdown of a chronically diseased heart and on the other hand, too much rest for milder grades of cardiac insufficiency and especially for cardiac disease with no evidence of present or impending breakdown.

In general, an acute carditis demands rest until the temperature and pulse are normal and until moderate exercise does not cause the temperature to rise, the pulse to show undue irritability, or dyspnea to make its appearance.

The patient with failing heart, whether from primary chronic valvular or myocardial lesion, needs rest long after his more urgent symptoms have disappeared. A too early resumption of work will promptly cause another breakdown—the old story familiar to every hospital doctor. So rest is proper, rest is preached to our students and practitioners; and our heart societies plead for convalescent homes for these two types of cases, the one recovering from an acute carditis and the other from an acute breakdown in the course of a chronic disease.

No wonder then that many doctors feel that prolonged rest is essential in all heart cases. They get the habit of prescribing it even though the heart is frankly competent, merely because some slight deviation from the normal has been found. They constantly emphasize the avoidance of exertion.

Many physicians need to have removed the bugbear fear of exertion. It is no new thought that the heart muscle after the subsidence of the acuter symptoms may, like skeletal muscles, need exercise; that what is needed in many instances is not less, but more exercise. Patients must be told that a rheumatic heart or a leaky valve does not necessarily imply a life of inactivity and idleness. Individualization is the key to this feature of treatment. We may not all agree with Dr. St. Lawrence, who says that with children, after an acute rheumatic endocarditis daily exercise to the point of tolerance is to be advised, but we must agree with his advocacy of placing a time limit to rest and substituting therefor a system of training of the heart muscle so that it may recover its tone and reach and maintain its maximum of efficiency.

And what about digitalis? I know I am treading on dangerous ground when I venture to say that there is a too strong tendency to impose upon the profession arbitrary rules as to the use of this drug, rules that are in part contraindicated by the every-day results of bedside practice. The fact that so many physicians successfully prescribe digitalis without thought of cat units, accurate body weight, or rate of elimination; that they use it in conditions other than auricular fibrillation; that they do not refrain in an urgent situation from giving it subcutaneously, intramuscularly or intravenously; that they very rarely feel compelled to give the massive dose in order to get the prompt effect,—all this is a protest—an unconscious one to be sure—against the too dogmatic directions that have been given publicity, directions that are perhaps too much based on laboratory experiment, with too little control of extensive bedside observation. The clinician often knows that he can get good results by the intramuscular or intravenous route when by the stomach he is disappointed. He finds at times that if he spares the stomach there may not be as much nausea though he does not dispute the statement that the nausea and vomiting of digitalis are primarily cardiac, and reflexly central, in origin. Instead of the massive dose, he may prefer for emergency effect the guarded intravenous use of strophanthin or ouabain. He thinks—sometimes he says he knows—that he gets results by giving digitalis in conditions other than fibrillation—in

hypertension with beginning failure, and even though blood pressure may be still high; in the failing heart of pneumonia and other infections; in valvular and myocardial lesions before fibrillation has set in. He may not be able to tell you why. He may not have a fixed dose. He gives till he gets results, which is the rule, we are told, that Cushney used to teach his students. In a similar manner he gives his morphin. He may try one-eighth or a sixth of a grain; or if the pain is severe as in renal colic, he may give one-third or even one-half grain. He is after results.

If timid, he may be fearful as to the toxic effects of digitalis. He knows, as Mr. Dooley said: "Drugs are a little iv a pizen that a little more would kill ye." So much has been said about cumulative and toxic effects—and with reason, for the danger from overdosing is a real one—that many doctors are deterred from using an efficient amount. Much too small and ineffectual doses are not infrequently seen. The good repute of digitalis is reestablished when there is an increase in the amount and favorable results follow.

The greatest fault, however, is the giving of digitalis without any indication for its use except the diagnosis of heart disease—and this diagnosis sometimes wrong.

It may be straying a little from the subject of treatment to refer to the fact that in many of these cases there has been a mistake in diagnosis that has led to the unnecessary use of digitalis. No heart lesion whatever, or an unimportant variation from the normal, has been regarded as pathological and as demanding treatment. A respiratory arrhythmia, an increased rate, a faint systolic murmur or a suspicion of one, a cardiac impulse a little too far to the left, a blood pressure a few points too high, a precordial uneasiness, these or other symptoms have led to the unwarranted diagnosis of heart disease, activity has been restricted, digitalis prescribed and our supposed cardiopath is on the way to become a confirmed neuropath. How does it happen? Ignorance, haste, carelessness, indifference, too great reliance on X-ray or electrocardiograph will explain many cases. But I believe the greater number are to be explained by the fact that the doctor has a surplus of honesty and is earnestly desirous of making an early diagnosis of heart disease. He has heard from teacher, he has read in the medical journal that the early diagnosis of heart disease as of tuberculosis or cancer, is the great desideratum. In this way and in this way only, can the serious cardiac breakdown be postponed.

But he has not read of the errors in the diagnosis of heart disease

and of tuberculosis. He should read what Edouard Rist has to say on this subject of errors in the diagnosis of pulmonary tuberculosis. Rist's figures, based largely on hurried wartime examinations, certainly must overstate the frequency of the error of more deliberate examinations in time of peace. Yet they are startling and illuminating. One-third of a large number of cases in France treated for tuberculosis were not tuberculosis at all. Rist quoted similar statistics for England. Friedrich von Mueller said a like error had been made in Germany. Heart disease and tuberculosis are not the same, yet the mistake of diagnosing wrongly may in either instance lead to direful consequences, unnecessary fear, lives of inactivity, enormous economic loss.

In this connection, a few words that have to do more with the treatment of the patient than of the disease.

There is ingrained in the minds of most people a greater fear of heart disease than of any other malady unless it be cancer or syphilis. To most laymen sudden or dropsical death is implied by the term heart disease. How important, therefore, that there be not aroused without reason, the thought of this condition. Yet how often the practitioner or the specialist as he examines the chest or gets the history, arouses the dread of something wrong with the heart, when perhaps there is nothing the matter. He may do it by a shrug of the shoulders, an air of hesitation or of perplexity as though not quite understanding what he hears, by a comment on a faint murmur or a skipped beat. He usually does this innocently, forgetting that it is not always wise to think out loud especially when there is an element of doubt or uncertainty and when there is present an interested yet apprehensive, impressionable listener.

Again when there is some real cardiac disturbance the greatest care is necessary not to convey a wrong impression. Emerson has said: "It is not the fact that imports but the effect of the impression of the fact on the mind." To tell a patient that he has a leaky valve or a large heart, i.e., that he has heart disease, may be to state a fact. Unless this statement be explained to suit the intelligence of the patient and to accord with the degree of severity of the heart lesion, a wrong impression may be left, the impression of a disease that is destined to advance relentlessly toward an early death. The statement, truthful as to fact, may be untruthful in its effects, i.e., its impression on the mind of the hearer. An unintentional lie has been uttered, as much an untruth as though the physician finding a definite organic disease flatly denies its existence.

There is a danger not fully recognized, that the movement for periodic health examinations will, unless most carefully watched, make people too introspective, too apprehensive. Unless the examinations are made by competent men, who understand human nature as well as disease and how to recognize it, greater harm than good may come. This will be not so much because doctors are ignorant or careless, as because they will feel the necessity of calling attention to slight departures from the normal that may really be of no moment, a procedure that may cause worry, anxiety or genuine nervous ill health in the susceptible individual. So too, our Heart Associations must guard against fostering needless fear of heart disease by paying too much attention to slight cardiac disturbances and unduly stressing the importance of early recognition of heart disease. We cannot afford to develop a national cardiac neurosis.

The plea in this paper is for greater care as to how we examine, how we tell our patients, how we prescribe rest and digitalis; a plea for individualization. There is no intent to undervalue the laboratory study of drugs. But the time is not yet at hand when we can quantitatively estimate the degree of cardiac failure or by reference to some slide rule, mechanically dose out rest or exercise or digitalis. The history, observation, physical examination, laboratory tests, and instruments of precision must all be given a place in the treatment of heart disease. Even psychology has a definite function.

"Now," said the cynic, after reading this paper, "did I not say the treatment was only rest and digitalis? That is practically what you have said."

"But," I replied, "I have emphasized the need of individualization, have suggested varying degrees of rest and the advantage of properly conducted examinations. To treat by a hard and fast rule of 'rest and digitalis' is unwise."

"Of course," pursued the objector, "I assumed that your practitioner or specialist was a man of experience and that he had common sense."

"Alas!" I replied, "some of us have knowledge but are not very wise; some of us see and know, but do not think logically; some of us have had experience but have not learned; some of us are lacking in good judgment or what you call common sense. The trouble, you see, is that we are all human. So, old, plain truths have to be repeated or presented in forms to fit the passing moment."

And that is my apology for a very simple, old-fashioned paper.

Congress Discussion.

DR. E. P. CARTER,

Baltimore.

I feel as if to the delightful remarks of the last speaker might be added as an epilogue the definition of a specialist—"as one who knows more and more about less and less."

We are all indebted to Dr. Swift and his associates for this exposition of their studies from which they have evolved their conception of the reaction of the heart to acute infections and especially the nature of the response in man to rheumatic infection.

I feel that I have had a surfeit of tremendously interesting and stimulating facts given me. The same questions that occur to me have, I am sure, occurred to many of you. It is only possible for me to follow Dr. Swift's explanation up to a certain point. It is difficult for us who have been largely influenced by tradition in our thoughts, particularly in regard to rheumatic infection, to dissociate ourselves from fixed ideas and to accept readily an entirely new hypothesis—an hypothesis which seems to suggest some very striking paradoxes.

Leaving out of consideration at this time syphilis and diphtheria, we must now consider rheumatic infection as a universal vascular tissue reaction of far-reaching significance. Dr. Swift has presented much sound evidence for a new conception of rheumatic infections and yet one that recalls certain similar suggestions in the older literature. I, personally, find it very difficult to correlate the widely divergent clinical picture of the course of the actual infection with the various streptococci with the cardiac response. There is an extraordinary dissimilarity of reaction found in the cardiac tissue in such clinically different infections as rheumatic disease and subacute bacterial endocarditis.

Without going into the difficult problems of immunology and allergy, it seems to me that the suggestive argument, which was voiced years ago, of the similarity of the reaction in tuberculosis and in syphilis to that in rheumatic infections, which we have heard commented upon, at least in their clinical manifestations, is of the greatest importance. I wonder, however, just how far we can link up the non-specific reaction of the tissues to the immunologically

different strains of streptococci recovered in these cases. The incidence of subacute bacterial endocarditis, occurring together with an acute rheumatic infection, is a very interesting and suggestive point. Further subacute bacterial endocarditis is so often implanted upon the previously diseased valve or endocardium which gives evidence of a previous rheumatic infection. It seems to me extraordinary that there is such a rarity of previous syphilitic infection in the cases of subacute bacterial endocarditis.

In order to crystallise in my own mind this question of the so-called allergic response of the tissues in rheumatic disease, I should like to ask Dr. Swift a number of questions. (1) What constitutes evidence of an allergic response on the part of the heart in any bacterial infection? (2) I should also like to ask where is the focus in man which gives rise to the sensitization of the individual as demonstrated by the intracutaneous inoculation? Is this focus in the throat, is it primarily in the myocardium, or in the valvulitis, which has been greatly stressed by Dr. Swift and his associates? (3) Again, is the infection of the myocardium a primary infection?

(4) We know that the Aschoff bodies represent a proliferative response on the part of the tissues, though we know nothing of their exact relationship to the infection. It may not be a proper question but I should like to ask what Dr. Swift considers may be the specific nature of the Aschoff body?

(5) One other point suggests itself. What may be the pathology of the bicuspid aortic valves of congenital origin not infrequently encountered and upon which we occasionally find engrafted a subacute bacterial endocarditis with streptococcus viridans? Does this congenital malformation represent a prenatal infection?

Again, quite far afield, but as another bit of comparative evidence. In the literature years ago some internist first suggested the relationship of periarteritis nodosa to rheumatic infection. I thought it was Kussmaul, but I am not certain. After seeing Dr. von Glahn's remarkable slides this morning the resemblance of the diffuse lesions to periarteritis nodosa suggested itself. It is of interest in this connection, though far removed from the significance of the myocardial reaction, that Gruber has recently emphasized the possibility of an allergic response as underlying the proliferative lesions seen in this condition.

It is possible that the two quite distinct paths of evidence, clinical and experimental, are gradually approaching each other. We have

had the evidence of a comparison between the two great infections, tuberculosis and syphilis and rheumatic disease marshalled with extraordinary precision; though I think that the basis of the evidence is largely as yet a matter of analogy. As I understand Dr. Swift, precipitins and agglutinins do not occur in the experimental animal infections. It is very difficult to interpret the widely different tissue response so unlike in the two infections—subacute bacterial endocarditis and rheumatic disease as related to the same or to non-specific streptococci. May I conclude by expressing my great appreciation of the stimulating interest aroused by Dr. Swift's presentation of his hypothesis.

DR. A. E. COHN,

New York.

This discussion forms a very singular and interesting landmark in connection with our ideas of chronic heart disease. If we look back at the opinions of 25 years ago, to the work of Strümpel, Osler and Krehl, heart disease was spoken of as having been due to rheumatic fever, scarlet fever, syphilis, lead and tobacco poisoning, and many other conditions without any effort having been made to discriminate between the forms of natural history of these different diseases. In the writings of that day there was a short chapter on etiology, a short chapter on pathological anatomy, a long chapter on the mechanism of the disturbed blood flow, and a long chapter on physical signs in reference to valvular disease. This shows the difference in the attitude of yesterday and to-day; it is a matter of judgment whether the change is good. Not only has Dr. Swift advanced the theory of an immune reaction in rheumatic fever, but he has shown that different agents are causing this and other heart diseases and also that the pathological anatomy is quite distinct and appropriate to each of the separate diseases. We have therefore differences in all three aspects—the etiological agent, the pathological anatomy and the natural history—all different, depending on the disease which is present.

Dr. Emerson has mentioned certain interesting factors involved in the various forms of heart disease according to the period of the life cycle in which they fall especially in connection with their etiology—rheumatic fever falls in the earlier decades; syphilis causes the heart lesions of middle life, while the arterio-sclerotic group falls in the

later decades. The incidence of these forms as regards age groups and their incidence in regard to involvement of different sections of the population differ apparently strikingly.

The new point of view requires also a new outlook in therapeutics. In 1900 a simple chapter was usually written in the text books, but now on the basis of differences in processes of these various diseases the study of therapy must move forward in the same direction as that of etiology and of natural history. This is an important advance.

In all of this it seems to me that I have missed the mention of one point of importance. It is a matter of common knowledge that of those who die of the heart diseases 1/6th die before the age of 40 and 5/6ths after this age. The suggestion is improbable that syphilis alone is responsible for this disproportion; other circumstances no doubt contribute to this state of affairs. It appears to be a natural consequence of the aging process. The human body dies as the result of the mechanism of old age, and this process should be taken into consideration as a factor contributing to cardiac death. If so many deaths occur in association with the process of senescence, this circumstance must naturally be considered in connection with prevention and prognosis.

Prevention plays an important rôle in the whole picture. These various points have been brought before us in a very eloquent fashion.

DR. GEORGE CANBY ROBINSON,

Nashville, Tenn.

I would like to discuss especially the question of heart strain. It is evident to all of us that the work of the heart is varied by many things all the time and an increased amount of work becomes a strain only when the heart fails to respond to the increased work in the normal way. There are many factors of safety in the circulation, and it is only when these factors are unable to meet the demand put upon the circulation that symptoms of heart disease appear. The signs of heart damage may be present without symptoms, a condition implying that the circulation is in a state of compensation.

The heart is an organ engaged in the distribution of blood, but after all it is primarily concerned in the distribution of oxygen to every cell of the body from the atmosphere in which we live. This is the primary function of the heart which it performs in coöperation with

other organs. I want to emphasize particularly the importance of the heart in relation to the respiration and to the vascular system. We have been interested in the mechanism of cardiac failure. In thinking of this matter we have to take into account a number of well-balanced mechanisms that have to work together to maintain the circulation in a state of efficiency and to keep the individual in health. The heart undergoes many strains and many forms of damage, and the remarkable thing is that it has the ability in the changes that take place to maintain the very accurate balance that is necessary for the respiration and circulation.

In studying the heart in heart failure and decompensation we must think of the heart not only as a single organ but also of its relations to other great physiological activities that are going on in the body and participating in the essential activity of distributing oxygen throughout the body. This point of view has received more attention in the recent investigations on the circulation, and less attention is being directed solely to the heart than when so much thought and study was being given to the cardiac arrhythmias. In the last few years studies have been pursued which have given means of analyzing further the circulation as a whole: studies of vital capacity, the study of the output of the heart per minute in man, and the studies of Blumgart on the velocity of the blood flow, have all broadened our conceptions of the circulation and are leading to a wider viewpoint.

The point I wish to emphasize is that in any consideration of heart strain and its consequences, it is necessary to bear in mind the various physiological activities with which the heart must coöperate in order to maintain effectively its functions.

DR. HENRY SEWALL,

Denver, Colo.

A general practitioner cannot come to the treatment of heart disease with better experience than the treatment of tuberculosis. I think we shall all agree that for that treatment we must have perception of the activity of the disease.

It may be inferred, speaking broadly, that it is not the anatomical limitations of an organ that render exercise dangerous, but that morbid chemical state which has been given the name *allergy,* in which inordinate response follows minimal specific stimuli, with intensification and extension of the disease focus. It is probably

true that an organ which is chemically normal, however mechanically crippled, fares best when furnished with a certain optimum of blood supply induced by exercise. More than twenty years ago Dr. Yandell Henderson, of Yale, published experiments upon the heart of a dog which are suggestive in this connection. He found, in brief, that with a slow rate of beat the diastolic volume of the heart increased but the systole was not complete, residual blood remained in the ventricle at the end of its contraction. *Pari passu* with rising frequency in the heartbeat the diastolic expansion of the ventricles became less and less and the completeness of systolic contraction more and more.

Now remembering that the coronary blood flow which nourishes the heart is directly related to the movements and the volume of the organ, it seems to me that we may assume better nutrition for the heart that is put through its paces as described. In Nature this is accomplished by the simple device of exercise. Medical experience abundantly testifies that in the organically "diseased" heart, with lesions compensated and inactive, these principles are best qualitatively, if not quantitatively, carried out as in normal life.

But follow that same heart back to the time of recovery from its first infection originating, say, in a pyogenic tonsil, the heart appears absolutely normal to examination. Another attack of tonsilitis supervenes and now soft murmurs can be heard at the apex. May we not suspect that an allergic organ has responded to a specific stimulus? Physical exercise a few days before the attack might have been highly salutary; now it would be hazardous in the extreme. The heart again becomes apparently normal; but again and again its allergic response is provoked and the signs of tissue destruction and attempts at repair gradually become permanently manifest on examination.

As in the case of tuberculosis, we can well suspect that the ultimate disaster is the effect of applying the right thing, exercise, at the wrong time, the period including and following acute infection.

DR. LOUIS FAUGERES BISHOP,

New York City.

I think this question of exercise and rest is very vital to the whole matter of heart disease. I am very much impressed by my own experience in practice, as to the importance of exercise to that group of people of middle age, who feel some precordial discomfort on

exertion and on that account consult the specialist. I want to make this assertion, that if one takes the whole group of middle-aged business men, showing signs of beginning degeneration symptoms of the heart, and divides them arbitrarily into two sections—to the first group give nitroglycerin, give them instructions to play golf, keep them out of doors, and make them keep up a moderate but regular exercise; take the second group of patients with identical symptoms, tell them to be careful, allow them to take unlimited rest and become lazy invalids—my experience has shown me that while the first group is alive and playing golf and keeping fit, the second group of patients is in line for first class funerals.

DR. S. A. LEVINE,

Boston.

I hesitate to ask a question of Dr. Herrick, especially if it infers a difference of opinion; because I have learned that his judgments are almost always sound. I would like to know, however, if he has any direct evidence that work improves the heart. I am very anxious indeed to learn of just this type of evidence. We all know that work produces a sense of well-being. Playing golf makes us feel better and happier, but analyzed purely from the point of view of the heart I am in doubt whether any good is done. I do not mean that we tell heart patients not to work but I do not know that work improves the heart. In fact I am inclined to the opposite opinion. We know that work aggravates the condition when patients have heart failure. If there is heart disease, rest in bed improves the circulation, possibly by re-establishing the balance between the two sides of the heart. The laws of nature are apt to be simple, and if it is true that rest improves diseased states it would be queer if the same organ were benefited by the opposite. Exercise does not improve the breathing capacity of the lungs; at least there is no evidence to show that this is so. In the case of long distance runners, men in the pink of physical condition, the vital capacity of the lungs is sometimes below normal and at others is normal or above normal. I made such observations some years ago in a group of marathon runners. I have felt that if exercise improves the breathing ability which is the best index of circulatory efficiency, these fellows should have had unusually large vital capacity readings, but they did not. In heart patients the vital capacity is diminished when failure is present, and

rises as improvement sets in. This is indirect evidence that exercise does not improve the ability to breathe. Expert runners run better because their legs are better.

Another point I would like to emphasize is this,—the conception that rheumatic fever is much more common than has been recognized. It might be regarded in the same light as infantile paralysis in which the number of cases affected is much greater than the number that have paralysis. There are many cases in children with an unexplained fever and vague symptoms in which there are no pains at all. The slight fever may last weeks, months, or years. Light may be thrown on the true nature of the underlying rheumatic condition in such cases by the secondary features that may be uncovered. In the family history we may find that a sister has heart disease, or a brother has chorea or rheumatic fever. Other important points are the repeated attacks of epistaxis and nausea and vomiting. These are peculiar symptoms that are somehow related to the rheumatic infection. They may occur before, during or after the apparent activity of the rheumatism. In other words there are many instances in children of slight fever, the finding of a slight systolic murmur and very little else which are due to rheumatism, in the entire absence of any pains or chorea. The question comes up whether any peculiar allergic factor decides whether the heart is going to be permanently involved or not. Many of these mild cases escape any serious cardiac damage, are regarded lightly as suffering from a benign or functional systolic murmur, and years later end up with a bacterial endocarditis. This point of view explains why so many patients who have mitral stenosis or more particularly bacterial endocarditis, give no previous history of outspoken rheumatic fever or chorea. I wonder if there is anything in Dr. Swift's work which throws light on the question as to which patients will and which will not end up with an organic lesion after a rheumatic infection.

DR. HOMER F. SWIFT,

New York.

(Closing.)

We recognize that some of the theory of the allergic nature of rheumatic fever rests on comparing analogous reactions in man and experimentally infected animals. A patient with rheumatic fever shows marked skin reaction following intracutaneous injection of

small amounts of killed streptococci, and tuberculin-like febrile reactions following intravenous inoculation. At times there are also focal cardiac reactions. There may be multiple foci where the allergizing substances are formed; e.g., diseased tonsils, sinusitis, abscesses at the apices of teeth, pneumonitis, and possibly areas in the gastrointestinal tract. The rapidity with which improvement sometimes occurs following tonsillectomy can be best explained as a result of the removal of such an allergizing focus; the failure to obtain permanent improvement may, on the other hand, be due to the existence of other foci. Bacterial products may be deposited at various places in the tissues, without any inflammatory reaction taking place until the entire animal becomes allergic; then marked inflammation occurs. It is impossible, as yet, to state the exact nature of the Aschoff body.

DR. PAUL D. WHITE,

Boston.

(Closing.)

I would like to thank Dr. Robinson for his discussion and especially for pointing out the correlation of the condition of the lungs and of the peripheral circulation to heart strain.

BUSINESS SESSION OF THE CONGRESS.

At the conclusion of the Scientific Session of the Congress a business session was held to consider a change in the interval between the sessions of the Congress. The Secretary read the following recommendation from the Executive Committee of the Congress. "(1) We, the Executive Committee of the Congress of American Physicians and Surgeons, recommend that no action be taken on the afternoon of May 2nd by the Congress and that the matter be referred back to the individual component associations for their discussion at their next annual meeting and that their votes be communicated to the Executive Committee of the Congress. (2) That the Executive Committee of the Congress be given authority to act in accordance with the vote of the component associations." Carried.

The Secretary then read a resolution prepared by Dr. Frank Billings of Chicago to be sent to Dr. William H. Carmalt of New Haven, who was unable to be present at this session.

"*To the Congress:* This Congress was organized in 1887—forty-two years ago. This is the fourteenth session of the Congress and like all the other meetings of the past is characterized by evidences of constructive work by all the constituent association members.

The administration of the Congress has been characterized uniformly by efficient energy and good judgment.

This standard of administration has been due in a large measure to the first secretary of the Congress, Dr. William Henry Carmalt of New Haven, Connecticut. Dr. Carmalt served as Secretary from 1887 until 1911—a period of twenty-four years. He was then made, and is still, the Chairman of the Executive Committee of the Council. For forty-two years Dr. Carmalt has been an executive officer of the Congress and his work has always been efficient.

Dr. Carmalt is now in his ninety-third year. Still vigorous mentally, his physical condition does not permit him to attend this Congress—the only one from which he has been absent in forty-one years. We can television this loyal man at his home, unhappy because he cannot be with us.

THEREFORE, We here in Congress assembled do herewith send Dr. William Henry Carmalt greeting—our hearty wishes for his health and happiness, with our esteem, our respect and our affection." *

It was then moved and seconded this be sent as from the Executive Committee. Carried.

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* Dr. Carmalt died July 17, 1929.

Another resolution was then read by Dr. C. W. Richardson.

"WHEREAS, in 1926, after a full hearing, Congress determined that the tax on physicians under the Harrison Narcotic Act should, in justice, be reduced from $3.00 to the pre-war tax of $1.00; and

WHEREAS, the Finance Committee of the Senate, on May 1, 1928, reported to the Senate a tax reduction bill increasing the tax of physicians under the Harrison Narcotic Act from $1.00 to $3.00; and

WHEREAS, there is no justification in the proposed increase and no opportunity was afforded physicians to be heard before the the Finance Committee in opposition to this increase,

Be it resolved, by the Congress of American Physicians and Surgeons, in session assembled in the City of Washington, May 2, 1928, that strong protest be entered against this unwarranted action on the part of the Senate Finance Committee and that the Congress of the United States be urged not to concur in the Recommendation of the Senate Finance Committee."

After a discussion by Drs. Darlington, Mercur and Alexander Lambert, this resolution, which was previously seconded, was withdrawn.

Adjournment.

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